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This report assesses the risk of exposure to benzene. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in March of 1981.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of benzene in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to benzene for various subpopulations.

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EPA-440/4-85-006
March 1981
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AN EXPOSURE AND RISK ASSESSMENT FOR BENZENE

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OFFICE OF WATER REGULATIONS AND STANDARDS
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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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EXECUTIVE CONCLUSIONS[1]

Excessive occupational exposure to benzene has been shown to be associated with leukemia, and other serious blood diseases. Acute toxicity involves central nervous system effects which can result in death.

Environmental exposures are at least 100 fold below the minimums thus far shown to be associated with toxicity from occupational exposures. However, prudent public health policy, by not recognizing thresholds for carcinogenic effects, would anticipate some risk at such levels. Conservatively applying the EPA Cancer Assessment Group's linear nonthreshold extrapolation to low exposures, the effect of benzene exposure routes on the potential cancer incidence in the total U.S. population would be estimated as shown below:

Comparison of Benzene Exposure Routes
(EPA Exposure and Risk Estimates)

Route	Average Benzene Level	Average Excess Lifetime Risk	Nationwide Incidence (cancers/year)
Ambient Air	3.3 - 6.5 $\mu g/m^3$	$2x10^{-5}$ - $5x10^{-5}$	75 - 150
Drinking Water	0.025 - 0.17 $\mu g/l$	$4x10^{-8}$ - $2x10^{-7}$	0.1 - 0.8
Food	possibly 250 μg/day	possibly greater than air	
Cigarettes	90 μg/cigarette	10^{-3}	800

The assumptions incorporated into the EPA cancer risk extrapolation suggest that these estimates may exceed the actual risks from the above tabulated exposure levels (as discussed in Chapters 5 and 7). The above estimates (or range of estimates) for the population mean do not reveal the distribution of individual exposures within the population. Individual exposures may be two orders of magnitude higher than the means, as described in Chapter 5. Nevertheless, these estimates provide some indication of the possible overall importance of benzene in the environment, since for linear nonthreshold risk extrapolations, incidence is determined by the arithmetic mean exposure. (The above estimated means and the Chapter 5 and 7 estimates for specific scenarios are intended to complement each other.)

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[1] Prepared by EPA Technical Project Officer based in part on program considerations.

It is apparent that cigarettes, food, and ambient air constitute the most important exposure routes for the non-occupationally exposed general population. Drinking water appears to comprise less than one percent of average exposure. Because the food data is very limited, the exposure via food is uncertain. While its presence at very low levels may represent a phase equilibrium with contaminated air, its presence at higher levels, such as in eggs, is believed to occur naturally. Due to benzene's low potential for bioconcentration, exposure to waterborne benzene via contaminated fish is expected to be less than via drinking water.

Relatively little risk to aquatic life can be expected to result from current environmental levels. Benzene is acutely toxic to some fish and aquatic invertebrates at concentrations above about 5000 µg/l. Although there is insufficient data to establish a chronic toxicity criterion, limited data suggest that chronic toxicity to fish may sometimes occur at concentrations in the range of 100-1000 µg/l. Of 185 ambient water measurements recorded in STORET, none exceed 1000 µg/l, and only 5 percent exceed 100 µg/l. No fish kills on file for the last decade have been attributed to benzene spills or discharges.

Although benzene is a naturally occurring substance, its global production and environmental burden have been increased by human activities. Approximately 11 million metric tons of benzene per year are handled within the U.S. economic system. One half of this is essentially pure benzene, mostly produced from petroleum by catalytic or thermal reactions, and used almost entirely as a feedstock to synthesize other chemicals. The other half is a constituent of hydrocarbon mixtures, primarily gasoline and other fuels.

Nearly all known environmental releases of benzene are to air, primarily from gasoline combustion. Less than one percent of the known releases is to water, primarily from solvent users, petroleum refiners, and chemical plants. Benzene disposal to land appears to be negligible; however, the content of some potentially important solid wastes is not known. It may be noted that the relative proportions of water and air disposal are very roughly equivalent to the relative proportions of average water and air exposures.

In soil the fate of benzene wastes is somewhat uncertain, and may involve volatilization, biodegradation, or leaching. In most surface waters volatilization is expected to dominate over degradation, thereby bringing benzene into the atmosphere, where it is oxidized. Water in equilibrium with contaminated urban air having 10 $\mu g/m^3$ benzene would have only 0.044 µg/l, and would represent negligible exposure compared to the air concentration. Nevertheless, such equilibrium may not be approached quickly, but may require a distance of a few miles to many dozens of miles, depending on a stream's depth and turbulence. The absence of substantial levels of benzene in ambient water is thus consistent with both the sparsity of discharges and the high fugacity of waterborne benzene.

Overall, it can be concluded from the assessment of benzene disposal, fate, exposure, and risk that:

1) Population aggregated exposure through waterborne routes (drinking water and eating fish) is small compared with exposure through either air, smoking, or possibly food.

2) Water discharges of benzene are small compared with air emissions, and thus, even when volatilized, do not substantially increase nationwide air concentrations.

3) Air contamination with benzene does not cause serious contamination of water, as through rainout.

4) The potential for aquatic life problems downstream of most benzene dischargers appears to be quite low.

5) Due to benzene's multi-media exposure potential, removal from one medium (such as water) by transfer to another (such as air) may not necessarily be of benefit.

Notes on Tabulated Cancer Risk Estimates:

1) Unit risk (dose-response) is taken from EPA (1980), referenced in Chapter 5. Other unit risk estimates are described in Chapter 5.

   a) Lifetime ingestion of 13.5 μg/day would result in 10^{-5} risk. Drinking water intake is assumed to be 2 l/day, although this may be high (Appendix C).

   b) Lifetime inhalation of 1.35 $\mu g/m^3$ with 50% absorption efficiency would result in 10^{-5} risk.

   c) Annual incidence is for the entire U.S. population (220 million persons), assuming a 70 year average lifespan.

2) Two estimates are provided for the air concentration averaged over the entire population. The lower is from Mara and Lee (1978), as referenced in Chapter 4; the higher is from Chapter 5.

3) The drinking water mean concentration is assumed to be represented by the National Organic Monitoring Survey. The range of estimates for the average was generated by assuming either:

   a) Benzene not detected implies zero concentrations;

   b) Benzene not detected implies a concentration just below the detection limit.

Two other EPA surveys support this general magnitude: the Community Water Supply Survey and the National Organic Surveillance Program (SRI).

4) The food exposure is an NCI estimate based on very little data (Chapter 5).

5) The cigarette smoking exposure is as described in Chapters 5 and 7. This risk is applicable to 54 million smokers.

TABLE OF CONTENTS

Page

EXECUTIVE CONCLUSIONS iii

LIST OF FIGURES vii

LIST OF TABLES ix

1.0 TECHNICAL SUMMARY 1-1

1.1 Risk Considerations 1-1
  1.1.1 Humans 1-1
  1.1.2 Biota 1-2
1.2 Materials Balance 1-2
1.3 Environmental Fate of Benzene 1-3
1.4 Environmental Monitoring of Benzene 1-5
  1.4.1 Human Effects and Exposure 1-6
  1.4.2 Biotic Effects and Exposure 1-9

2.0 INTRODUCTION 2-1
References 2-3

3.0 MATERIALS BALANCE 3-1

3.1 Introduction and Methodology 3-1
3.2 Production of Benzene 3-3
  3.2.1 Direct Production from Oil 3-3
  3.2.2 Direct Production from Coal 3-8
3.3 Imports and Exports of Benzene 3-10
3.4 Indirect Sources of Benzene 3-11
  3.4.1 Coal Coking 3-11
  3.4.2 Petroleum Refining for Gasoline 3-12
  3.4.3 Use of Products Contaminated with Benzene 3-12
  3.4.4 Natural Gas Well Condensates 3-14
  3.4.5 Resource Mining and Processing Operations 3-14
  3.4.6 Benzene Releases from Oil Well Drilling 3-14
  3.4.7 Benzene Releases from Oil Spills 3-17
  3.4.8 Combustion of Petroleum-based Fuels 3-17
    3.4.8.1 Benzene in Gasoline 3-17
    3.4.8.2 Benzene in Other Petroleum-based Fuels 3-20
3.5 Use of Benzene 3-20
  3.5.1 Consumptive Use 3-20
  3.5.2 Nonconsumptive Use 3-24
    3.5.2.1 Solvent Use 3-24
    3.5.2.2 Pesticide Use 3-26
3.6 Transportation and Storage of Benzene 3-26
3.7 Summary 3-27
References 3-31

TABLE OF CONTENTS (Continued)

Page

4.0 FATE AND DISTRIBUTION OF BENZENE IN THE ENVIRONMENT 4-1

4.1 Introduction 4-1
4.2 Physical, Chemical, and Biological Characteristics of Benzene 4-1
4.3 Monitored Levels in the Environment 4-3
  4.3.1 Air 4-3
  4.3.2 Water 4-5
    4.3.2.1 Drinking Water 4-8
    4.3.2.2 Ambient Water 4-8
  4.3.3 Soil 4-14
  4.3.4 Food 4-14
  4.3.5 Summary 4-14
4.4 Environmental Fate Modeling 4-14
  4.4.1 Equilibrium Partitioning 4-14
  4.4.2 EXAMS Modeling 4-22
  4.4.3 Intermedia Transfers 4-27
    4.4.3.1 From Air Medium to Surface Waters or Land 4-27
    4.4.3.2 Intermedia Transfers from Water Medium 4-31
    4.4.3.3 Intermedia Transfers from Soil Medium 4-33
  4.4.4 Intramedia Fate Processes 4-36
    4.4.4.1 Air 4-36
    4.4.4.2 Water 4-45
    4.4.4.3 Soil 4-50
    4.4.4.4 Plants 4-52
4.5 Summary 4-52
  4.5.1 Intermedium Transfer Processes 4-53
    4.5.1.1 Air 4-53
    4.5.1.2 Water 4-53
    4.5.1.3 Soil 4-53
  4.5.2 Intramedium Fate Processes 4-55
    4.5.2.1 Air 4-55
    4.5.2.2 Water 4-55
    4.5.2.3 Soil 4-55
  4.5.3 Critical Pathways for Specific Sources of Benzene 4-55
References 4-57

5.0 HUMAN EFFECTS AND EXPOSURE 5-1

5.1 Human Effects 5-1
  5.1.1 Pharmacokinetics 5-1
    5.1.1.1 Absorption 5-1
    5.1.1.2 Distribution 5-2
    5.1.1.3 Metabolism and Excretion 5-7

TABLE OF CONTENTS (Continued)

Page

5.1.2 Acute Effects 5-10
5.1.3 Chronic Effects 5-10
5.1.3.1 Pancytopenia 5-10
5.1.3.2 Leukemia 5-14
5.1.4 Summary of Effects on Humans 5-18
5.1.4.1 Ambient Water Quality Criteria -- Human Health 5-18
5.1.4.2 Additional Health Effects 5-21
5.1.5 Estimated Dose/Response Relationship for Cancer 5-22
5.1.6 Discussion of Available Data 5-24
5.1.6.1 Infante Study 5-24
5.1.6.2 Aksoy Study 5-26
5.1.7 Application of Dose/Response Models to Estimation of Human Risk - 5-26
5.2 Human Exposure 5-31
5.2.1 Introduction 5-31
5.2.1.1 Populations Exposed through Contaminated Drinking Water and Foodstuffs 5-31
5.2.1.2 Population Exposed through Inhalation 5-34
5.2.1.3 Percutaneous Exposure 5-35
5.2.2 Comprehensive Exposure Scenarios 5-37
5.2.3 Summary 5-40
References 5-41

6.0 BIOTIC EFFECTS AND EXPOSURE 6-1

6.1 Effects on Biota 6-1
6.1.1 Introduction 6-1
6.1.2 Mechanisms of Toxicity 6-1
6.1.3 Freshwater Organisms 6-2
6.1.4 Marine Organisms 6-7
6.1.5 Factors Affecting the Toxicity of Benzene 6-8
6.1.6 Conclusions 6-8
6.2 Exposure of Biota to Benzene 6-10
6.2.1. Exposure Route 6-10
6.2.2 Fish Kills 6-10
6.2.3 Monitoring Data 6-11
6.2.4 Exposure 6-11
References 6-14

7.0 RISK ESTIMATES FOR BENZENE EXPOSURE 7-1

7.1 Humans 7-1
7.2 Biota 7-3

TABLE OF CONTENTS (Continued)

Page

APPENDIX A. Vehicle Release of Benzene A-1

APPENDIX B. EXAMS Scenarios B-1

APPENDIX C. Liquid Consumption for Exposure Estimates C-1

LIST OF FIGURES

Figure No.		Page
1-1	Summary of Benzene Materials Balance, 1978	1-4
3-1	Producers of Benzene from Petroleum and Coal, 1978	3-6
3-2	Materials Balance for Benzene in Gasoline	3-19
3-3	Materials Balance for Benzene	3-29
4-1	Distribution of Unremarked Benzene Concentrations by Ambient Water Quality Station, 1977-81	4-9
4-2	Distribution of Remarked Benzene Concentrations by Ambient Water Quality Monitoring Stations	4-9
4-3	Schematic of Environmental Compartments Selected for Estimation of Equilibrium Partitioning of Benzene	4-20
4-4	Results of EXAMS Modeling of the Environmental Fate of Benzene in a Turbid River	4-23
4-5	Results of EXAMS Modeling of the Environmental Fate of Benzene in a Clean River	4-24
4-6	Results of EXAMS Modeling of the Environmental Fate of Benzene in an Oligotrophic Lake	4-25
4-7	Results of EXAMS Modeling of the Environmental Fate of Benzene in a Eutrophic Lake	4-26
4-8	Percentage of Benzene Reduction at Downstream Distance from Aquatic Discharges	4-34
4-9	Dispersion Modeling Results for Each Type of Source Category	4-39
4-10	Degradation Pathways for Benzene	4-47
4-11	Major Fate Preocesses for Benzene	4-54
4-12	Critical Pathways for Benzene (Released Amounts for 1978 Materials Balance)	4-56
5-1	Metabolic Pathway of Benzene in Liver	5-8

LIST OF TABLES

Table No.		Page
1-1	Comprehensive Exposure Scenarios for Benzene	1-8
3-1	Materials Balance for Benzene, 1978	3-2
3-2	Producers and Production of Benzene, 1975-79	3-4
3-3	Benzene Releases from Direct Petroleum Production, 1978	3-7
3-4	Producers of Benzene from Coke	3-9
3-5	Petroleum Industry Disposal of Solid Wastes Containing Benzene	3-13
3-6	Benzene in Contaminated Solvents	3-15
3-7	Gross Annual Discharges of Benzene to Water from Resource Mining and Processing, 1976	3-16
3-8	Estimated Benzene Content of Fuels	3-21
3-9	Summary of Consumptive Uses of Benzene, 1978	3-22
3-10	Materials Balance for Benzene in Chemical Feedstocks	3-23
3-11	Estimated Air Releases of Benzene from Use as a Solvent, 1978	3-25
3-12	Summary of Annual Environmental Releases of Benzene	3-28
4-1	Properties of Benzene Related to Environmental Distribution	4-2
4-2	Levels of Benzene in Air	4-4
4-3	Levels of Benzene in Air Near Chemical Plants and Petroleum Refineries	4-6
4-4	Levels of Benzene in Air for Human Activities	4-7
4-5	Concentrations of Benzene by Major Basin in 1980	4-10
4-6	Levels of Benzene in Water Near and In Effluents From Chemical Plants	4-12
4-7	Levels of Benzene in POTW Sampling Data	4-13

LIST OF TABLES (Continued)

Table No.		Page
4-8	Levels of Benzene in Influents to Sewage Treatment Plants	4-15
4-9	Levels of Benzene in Soil near Chemical Plants	4-16
4-10	Foods Reported to Contain Benzene	4-17
4-11	Values of the Parameters Used for Level I Calculation of Equilibrium Concentrations of Benzene Using MacKay's Fugacity Method	4-19
4-12	Equilibrium Partitioning of Benzene Calculated Using MacKay's Fugacity Method	4-21
4-13	Half-lives for Transformation and Transport of Benzene for Several EXAMS Scenarios	4-28
4-14	Exposure Analysis Summary for Benzene from EXAMS Model Runs	4-29
4-15	Rough Estimates of Ambient Ground-level Benzene Concentrations (8-Hour) Average Per 100 g/s Emission Rate from a Chemical Manufacturing Plant	4-38
4-16	Rough Estimates of 8-Hour Worst Case Benzene Concentrations Per 100 g/s Emission Rate Using the PAL Dispersion Model for a Chemical Manufacturing Plant	4-41
4-17	Concentrations of Hydroxyl Radicals and Ozone in Atmosphere of Different Environmental Settings	4-42
4-18	Oxidation Rate Constants and Half-lives of Benzene in Different Environmental Settings	4-42
4-19	Half-Life of Benzene in the Lower Troposphere	4-44
4-20	Benzene Biodegradation Rates	4-48
4-21	Microbial Species Isolated from Soil Capable of Degrading Benzene	4-51

LIST OF TABLES (Continued)

Table No.		Page
5-1	Estimates of the Saturation Half-life of Toluene and Benzene between Blood and Tissue	5-4
5-2	Toluene Concentrations in Air and Blood	5-6
5-3	Summary of Benzene Exposure and Related Hematotoxicity	5-11
5-4	Leucopenic Effects of Benzene	5-13
5-5	Carcinogenicity of Benzene in Experimental Animals	5-20
5-6	Comparison of Input Data for Calculation of Risk of Leukemia from Benzene Exposure	5-25
5-7	Predicted Excess Lifetime Leukemias per Million Population due to Benzene Ingestion (Inhalation), Based on the Study of Infante and Coworkers	5-27
5-8	Predicted Excess Lifetime Leukemias per Million Population due to Benzene Ingestion (Inhalation), Based on the Study of Aksoy and Coworkers	5-28
5-9	Estimated Benzene Exposure through Ingestion	5-33
5-10	Estimated Benzene Exposure through Inhalation	5-36
5-11	Summary of Estimated Benzene Exposure and Routes	5-38
5-12	Comprehensive Exposure Scenarios for Benzene	5-39
6-1	Effect of Benzene on Aquatic Flora	6-3
6-2	Acute Toxicity of Benzene to Invertebrates	6-4
6-3	Toxicity of Benzene to Freshwater Organisms	6-5
6-4	Toxicity of Benzene to Saltwater Organisms	6-6
7-1	Potential Risk Estimates for Benzene Exposure Scenarios Using Different Models	7-2
C-1	U.S. Beverage Consumption in 1979	C-2

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1.0 TECHNICAL SUMMARY

The Monitoring and Data Support Division, Office of Water Regulations and Standards of the U.S. Environmental Protection Agency is conducting risk assessments for pollutants which may enter and traverse the environment thereby leading to exposure to humans and other biota. The program is in response to Paragraph 12 of the NRDC Consent Decree. This report is a risk assessment for benzene using available data and quantitative models were possible to evaluate overall risk.

1.1 RISK CONSIDERATIONS

1.1.1 Humans

To assess the risks associated with the production and use of benzene to various human subpopulations, the results of exposure analysis for three comprehensive exposure scenarios were coupled with a series of mathematical risk models. Despite predominantly negative carcinogenic data for studies with laboratory animals, evidence that benzene is a probable leukemogen for man is convincing. Risk estimates were therefore based on human epidemiological studies of occupational situations.

Four risk extrapolation models were applied to the dose-response data to indicate the range in the predicted number of possible excess cases of leukemia that might result from chronic human exposure to benzene. The range of predicted risk obtained for the human exposure levels of interest is indicative of the inherent uncertainty associated with the mathematical models currently used for risk extrapolation purposes. There is presently no scientific concensus for selecting the most appropriate model for extrapolating high exposure levels associated with occupational exposure. Each of the models is formulated in such a way that the curves pass through the origin; that is, some finite response can be predicted at doses greater than zero. The no-threshold concept is scientifically debatable; however, it has been the position of some scientists and of government regulators that thresholds to carcinogens do not exist. By taking this position, the predicted risks tend to be "conservative," i.e., an overstatement of the risk.

The range of potential excess cases of leukemia predicted by the four mathematical models applied is described below for each of the three scenarios. The total human dose was computed on a milligrams per day basis, assuming that the response is dependent on absorbed dose but is independent of exposure route.

- Scenario A. Urban/suburban exposure (includes inhalation of typical urban air, food and drinking water ingestion and gas station use) combined with risk models, yielded a range of 5 to 560 potential excess leukemias per million people exposed.

- Scenario B. Rural/remote exposure (includes inhalation of typical rural air; food and water ingestion and gas station use) combined with risk models, yielded a range of 3 to 420 potential excess leukemias per million population exposed.

- Scenario C. Exposures near user/manufacture sites (includes inhalation of air with industrial scale benzene levels, and includes food and water ingestion and gas station use, as above) combined with risk models yielded a range of 21 to 1119 potential excess leukemias per one million people exposed.

It is noteworthy that drinking water appears to contribute less than 1% to the total exposures that result in the range of potential excess lifetime leukemias cited above. If one excludes residence near a user/manufacturing site, consumption of contaminated food appears to account for more than one-half of the total exposure (and thus of the risk) in the above three scenarios. Some uncertainty exists, however, in the food exposure pathway. Available data are incomplete; in the absence of data, it was necessary to assume that reported concentrations in certain foods were representative of all food groups utilized in estimating total intake. It is unknown how representative the reported concentrations are for foods in general. Exposure via gas station usage and inhalation of ambient air levels account for the balance of total benzene intake in the three exposure scenarios examined.

Additional risks of 3240 to 106,000 potential excess leukemias could exist for the 54 million people that smoke (i.e. 60 to 1960 per 10^6 population exposed), based upon 50% absorption of 90 ug benzene per cigarette, the consumption of 1.6 packs per day and the use of the models described above.

1.1.2 Biota

Biotic risks from benzene exposure could result from such events as spills of the chemical or gasoline. Despite the number of such spills, however, no fish kills have been reported. Ambient benzene levels are generally below reported effects levels; and, in the case of more concentrated effluent discharges, disturbances would occur to local populations only, rather than on a large-scale ecological basis.

1.2 MATERIALS BALANCE

The materials balance of benzene is somewhat unusual in that nearly equivalent quantities originate from "indirect" as well as from "direct" sources. Direct sources are petroleum refineries and coke plants; in 1978, including imports and inventory withdrawals, direct sources totaled 5,451,100 kkg. Indirect benzene sources include gasoline and other petroleum fuel refining, distribution and use, use of solvents contaminated with benzene, coal coking, and mining and resource processing. These sources totaled 5,754,600 kkg of benzene in 1978. Environmental releases resulting from these source operations were 27,000 kkg.

The major use of benzene is as a chemical feedstock. This amounts to 5,251,000 kkg. A small volume is exported (151,000 kkg) and only 9600 kkg are used directly as a solvent. Benzene is included in gasoline (4,665,800) and other fuels (959,400 kkg), and in other solvents (22,600 kkg). The environmental releases resulting from the use of benzene (including losses during transport and storage) were 246,100 kkg. Chemical transformations and fuel combustion accounted for destruction of 10,739,200 kkg. The overall materials balance (see Figure 1-1) had a discrepancy of only 1.0% of the total available benzene. (All materials balance data given are for 1978).

The materials balance developed for benzene using 1978 production and use figures shows total environmental releases of 246,100 kkg. Of this amount, 95.5% was emitted to air, 0.5% was discharged to water and 0.2% was land disposed (3.8% of releases could not be assigned to a specific medium). The largest source (72%) of benzene emission was the combustion of gasoline and other fuels. Transport and storage of gasoline, petroleum refining, and the use of benzene as a chemical feedstock also led to significant (21%) air emissions. Chemical production using benzene feedstocks and refinery production of benzene accounted for 76% of water discharges, to which petroleum refining contributed 94% of land discharges.

1.3 ENVIRONMENTAL FATE OF BENZENE

Benzene is a moderately volatile organic chemical with a relatively high water solubility, and a low chemical reactivity because of its stable ring structure. The environmental fate of benzene has been analyzed for inter- and intra-medium processes.

The most significant intermedia fate process is volatilization from either water or soil to air. Of limited overall importance are: rainout from air to soil or water, soil adsorption from water or desorption into water from soil and surficial runoff to water. Within the air medium, the dominant fate process is oxidation by hydroxyl radicals. Both soil and water biodegradation by microbial species may be important in some habitats, however, it is not universally important.

Thus, the three critical pathways that determine the ultimate fate of benzene released to the environment and act to reduce the total environmental benzene load are:

- Atmospheric sources (95.5% of total) → oxidative destruction.
- Aquatic sources (0.5%) → volatilization → oxidative destruction.
- Land sources (0.2%) → volatilization → oxidative destruction.

SOURCES OF BENZENE / USES

Direct	Indirect	Uses
Refineries	Gasoline Refining & Imports	Chemical Feedstock
Coke Plants	Other Fuels	Solvents
Imports	Coal Coking	Exports
Inventories	Contaminated Solvents	Gas and Fuel Consumption
	Oil Spills	
	Resource Mining/Processing	
5,451,100 kkg	5,574,600 kkg	11,143,800 kkg

TOTAL SOURCES = 11,025,700 kkg

Total Sources-Source Releases-Uses = Source Discrepancy

11,025,700 - 27,000 - 11,143,800 =

Source Discrepancy = -145,100

ENVIRONMENTAL RELEASES

From Direct Sources	From Indirect Sources	From Uses
4700 kkg	22,300 kkg	245,500 kkg

Direct Source Releases + Indirect Source Releases = Total Source Releases

4700 + 22,300 =

Total Source Releases = 27,000 kkg (see above)

Total Environmental Releases = 272,500 kkg

FIGURE 1-1 SUMMARY OF BENZENE MATERIALS BALANCE, 1978

Oxidative destruction has a short half-life (<4 days in urban settings) as does volatilization from water (~3 days by EXAMS model). Although no half-life is available for volatilization from soil, this process proceeds rapidly. Some portion of the amounts released will remain within the initial media as ambient levels until either biodegradation or entrance to a critical pathway occurs.

In summary, the bulk of benzene releases occurs in the one medium in which they are most speedily broken down and this destruction is the ultimate fate of most of the released benzene. However, rate limits to the oxidative destruction and to the intermedia transfer processes do act along with benzene's basic chemical properties to retain some benzene in water that is adsorbed onto sediments or to remain airborne.

1.4 ENVIRONMENTAL MONITORING OF BENZENE

Data have been collected for benzene concentrations in water, air, soil, and foodstuffs. Because of the traditional concern over benzene inhalation, especially in occupational situations, the most extensive data base covers air levels.

Air levels are typically between 1 and 3.5 $\mu g/m^3$ in areas remote from sources and between 4 and 160 $\mu g/m^3$ in urban areas where the number of sources (cars and industrial plants) is high, compared with 4.5 $\mu g/m^3$, which is a usual level in more residential areas. Atmospheric levels have been directly correlated with traffic volumes. Service stations are cited as a major source of benzene releases, with the levels for both rural and urban stations in the 1-32 $\mu g/m^3$ range. The levels near chemical plants have been as high as 824 $\mu g/m^3$, however, they are more typically around 14 $\mu g/m^3$ for chemical plants and 9 $\mu g/m^3$ at refineries.

The few data on benzene in drinking water indicate a median level of less than 2 µg/l for those samples that tested positively; to be conservative, a level of 2 µg/l was taken as a representative level of benzene in drinking water. For both food and water, the term "conservative" implies a higher level than may actually occur.

Most ambient and effluent levels in surface waters fell between 0 and 10 µg/l. However, the mean concentrations (104-638 µg/l) in Missouri, Kansas, and Michigan were as much as seven times higher than in other areas. The high values were generally reported in the vicinities of chemical plants and refineries. Benzene levels in raw wastewater were between <1 and 143 µg/l; removal averaged about 90%, with 3 of 5 plants achieving 100% removal. These data and sludge concentrations indicate that benzene is volatilized during aeration; biodegradation may also take place to a limited extent.

Few soil data were available. Levels between 13 and 115 µg/kg were reported in samples taken near chemical plants producing or using benzene.

Benzene has been detected in fruits, nuts, vegetables, dairy products, meat, poultry, eggs, fish and several beverages. Only a few of these items have been quantitatively analyzed and it is hypothesized that benzene may even be a naturally-occurring flavor component. Eggs have the highest documented levels (500-1900 µg/kg), followed by haddock (100-200 µg/kg) and Jamaican Rum (120 µg/kg). Cooked meats have levels of less than 10 µg/kg higher than those levels found in raw meats.

1.4.1 Human Effects and Exposure

Benzene is readily absorbed by all routes of exposure, with the rate of absorption dependent on both concentration and membrane permeability. Absorption via the lungs is rapid; most reports indicate a respiratory retention in humans of approximately 50% of inhaled benzene. The average skin permeability to benzene vapor is estimated to be 0.002 $m^3/(m^2\text{-hr})$. Dermal absorption of liquid benzene is much faster than that for vapor; the estimated absorption rate for liquid benzene through skin is 550 $mg/m^2/hr$.

Once absorbed, benzene is widely distributed to all tissues, with the rate of uptake by a tissue determined by the relative blood perfusion of that tissue. A large fraction of absorbed benzene is excreted unchanged in expired air, with the actual proportion dependent on dose and species. Estimates of the fraction excreted by humans range from 12 to 50%. Metabolic conversion of retained benzene occurs predominantly in the liver. The major metabolites include phenol, catechol and their sulfo- and glucuronic conjugates.

Single exposures to high levels (64,000 mg/m^3) of benzene are lethal within 5 to 10 minutes for man. Severe but non-fatal acute exposures produce headache, nausea, a staggering gait, paralysis and convulsions. Less severe exposures may produce giddiness and euphoria.

The relationship between chronic human exposure to benzene and hematological disorders, most notably pancytopenia and leukemia, has been extensively documented in the literature. Despite predominantly negative animal carcinogenicity data, there is strong evidence to suggest that benzene is a probable leukemogen in humans. However, case reports of benzene-associated leukemia generally relate to occupational exposures in industries where workers were exposed to rather high benzene concentrations (300-1200 mg/m^3) sufficient in themselves to produce pancytopenia and its variants. Pancytopenia, a deficiency of all cellular elements of the blood, in its most severe form, is a result of aplastic anemia. Furthermore, in any of the reported cases of benzene associated leukemia, occupational exposures to other organic solvents occurred concurrently with benzene exposure.

A dose-effect relationship between exposure and the incidence of these diseases is more difficult to establish due to uncertainties of occupational exposures. Several assumptions of considerable scientific debate are required. Most notably, an equivalence between relatively short-term, high-level occupational exposures and lifetime, low-level exposures was assumed. It is generally agreed that, while not ideal, this approach is conservative (i.e., it may overestimate the risk of leukemia to the general population resulting from chronic, low-level benzene exposure).

The U.S. EPA has estimated that a lifetime exposure to an ambient water concentration of 0.66 µg benzene/l will result in one additional case of cancer per million population exposed.

Human exposure to benzene has been analyzed for all routes because monitoring data indicate measurable levels in water, food, and air. Benzene levels for various exposure scenarios were analyzed to develop mean exposures. Exposure to benzene through drinking water may occur on a limited basis across the United States. Because only a fraction of the water supplies tested contained benzene, no distinction could be drawn between the potential exposures from ground versus surface waters. Using the conservative estimate of 2 µg/l, up to 4 µg/day could be ingested. Although some studies have found benzene in foodstuffs, the extent of contamination of food supplies, in general, is not known. The National Cancer Institute suggests 250 µg/day as a conservative (high) estimate.

The data base for inhalation exposure was broader, allowing the development of a larger number of scenarios. In urban areas, inhalation of background levels could typically contribute 0.1 mg/day, in suburban areas 0.05 mg/day, while 0.03 mg/day would be more usual in rural/remote areas. If subpopulations lived or worked near a refinery, 0.5 mg/day could be the resulting exposure. Residence or labor near chemical plants could contribute 0.01 mg/day. Gasoline stations, often the site of uncontrolled benzene emissions, have the potential to add 0.01 mg/day. Cigarette smoking has been documented as a possibly significant source of benzene, adding 1.4 mg/day for the average smoker. Occupational activities could add between 0.06-150 mg/day to the basic exposures listed above.

Dermal or percutaneous exposure in the home could be as high as 1.0 mg/use of a substance containing 5% benzene, with such exposures occurring sporadically. In an occupational setting, percutaneous exposures could add as much as 40 mg/day.

The exposures for the separate routes were added to develop three general, comprehensive scenarios (A, B, and C) and are shown in Table 1-1. Scenario A includes the largest population group (urban and suburban dwellers, 74% of the 1970 census population) and involves the appropriate inhalation levels, gasoline station usage on a periodic basis, and the basic food and water consumption. (By smoking cigarettes, 1.4 mg/day may be added to any of these scenarios.) Total average daily exposure is about 0.4 mg/day. The second largest population category, Scenario B, is rural dwellers, and has an associated total daily exposure of about 0.3 mg/day. A more rarely encountered scenario,

TABLE 1-1. COMPREHENSIVE EXPOSURE SCENARIOS FOR BENZENE

Route	Exposure by Scenario in mg/day		
	A	B	C
% of 1970 population	74	26	?
Ingestion			
Water	0.004	0.004	0.004
Food	0.25	0.25	0.25
Inhalation			
Baseline	0.1 (urban)	0.03 (rural)	0.5 (near sources)
Gas Stations	0.01	0.01	0.01
Cigarettes[a]	1.4[b]	1.4[b]	1.4[b]
Percutaneous			
Residential	<1.0[b]	<1.0[b]	<1.0[b]
Total Typical Exposure[c]	0.4	0.3	0.8
Potential Maximum[d]	2.8	2.7	3.2

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[a] A 1978, population of 54 million individuals who smoked cigarettes.

[b] This amount not included in total exposure.

[c] Excludes percutaneous exposure and that due to cigarette smoking.

[d] These amounts include all possible routes of exposure but not possible percutaneous absorption.

Source: This report.

C, would add exposure due to user site emissions for a total of up to 0.8 mg/day. These total typical exposures may be exacerbated by smoking, or home use of solvents or other benzene-contaminated substances.

1.4.2 Biotic Effects and Exposure

The lowest concentration of benzene at which effects have been observed in aquatic organisms is 0.001 mg/l, which affected growth in several algae species. Acute and sublethal effects to adult fish, including trout, bass and herring, were observed at levels <20 mg/l. Between 20 and 36 mg/l, several freshwater fish exhibited acute toxic effects. Algal growth was usually inhibited by benzene concentrations starting between 36 and 100 mg/l. Several more resistant species exhibited chronic and acute toxic effects in the 100-400 mg/l range. The most resistant algae species (Chlorella) showed inhibited growth and a reduced photosynthesis:respiration ratio in the 400-1755 mg/l range.

The levels of benzene that cause deleterious effects in aquatic biota are lower than some of the monitored water levels; thus some sub-lethal, but not serious effects may be expected. Benzene concentrations in refinery and chemical plant effluents were in the 0.08-1.0 mg/l range, while most ambient levels were <0.01 mg/l. The available data are far from comprehensive; therefore, it is difficult to accurately represent the total picture of aquatic contamination. However, these levels are generally lower by a factor of 2000 than those determined to be toxic.

The higher concentrations associated with chemical plants are also less (by 100x) than toxic levels for freshwater algae and fish. Overall, presently known ambient levels do not give rise to predictions of exposures of concern for acute or chronic effects for aquatic organisms.

2.0 INTRODUCTION

The Office of Water Planning and Standards, Monitoring and Data Support Division of the Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated included potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and estimate exposure of receptors to these substances. The results are intended to serve as a basis for developing suitable regulatory strategy for reducing the risk, if such action is indicated.

This document is an assessment of the exposures and risks associated with benzene in the natural and human environments. It includes summaries of comprehensive reviews of the production, use, distribution, fate, effects and exposure to benzene and the integration of this material into an analysis of risk.

Benzene is an aromatic, volatile, colorless liquid extracted primarily from petroleum and to a lesser extent from coal. Use as well as derivation of petroleum and petroleum products (i.e., gasoline and solvents) result in environmental releases of benzene as it may be a contaminant. Benzene is used predominantly as a chemical feedstock and is shipped throughout the country to many chemical plants. It is also used as an industrial solvent.

The physical and chemical behavior of benzene is described in Chapter 5.0. Its properties are documented in the first section, followed by a compilation of monitoring data for all environmental media. The results of media-specific fate and intermedia transfer models used to predict concentration levels of benzene in the air and water within close proximity to significant benzene sources and equilibrium concentrations resulting from free exchange of benzene between air, soil, water, and sediment are presented next. A discussion of biodegradation of benzene and a summary of critical fate pathways conclude the chapter.

The most current research of effects upon human and nonhuman receptors, a description of the duration of exposure and the populations exposed to documented or predicted levels of benzene and a statement of risk comprise the later chapters of this report.

Presently, there are two sets of federal criteria for benzene: occupational air standards and water quality criteria. In water, a concentration of 6.6 μg/l is associated with a 10^{-5} risk of human cancer (U.S. EPA 1980).

The Occupational Safety and Health Administration has established permanent standards for the regulation of benzene in the workplace. The time-weighted average concentration for 8 hours should not exceed 32 mg/m^3 (10 ppm), with a peak concentration of 160 mg/m^3 (50 ppm) for any 15-minute period during the 8-hour day (RTECS 1980).

Benzene concentrations in air were converted from ppm to mg/m^3 by using the following relationship: 1 mg/m^3 = 3.192 ppm. This factor was derived assuming 1 atm and 25°C, conditions, which were not absolute for all atmospheric measurements. However, monitoring data are seldom reported with the concurrent temperature and pressure; therefore, in the absence of these data, the conversion factor was used for all values of benzene in air.

REFERENCES

Registry of Toxic Effects of Chemical Substances (RTECS), 1979 Edition. Cincinnati, OH: U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health; 1980.

U.S. Environmental Protection Agency (U.S. EPA). Ambient water quality criteria for benzene. Washington, DC: Office of Water Regulations and Standards, U.S. Environmental Protection Agency; 1980.

3.0 MATERIALS BALANCE

3.1 INTRODUCTION AND METHODOLOGY

This chapter presents data on the production and consumption of benzene as well as data on releases of benzene to the environment. In April 1980, JRB Associates, Inc. prepared a report for use in this exposure assessment. Much of the data and text are drawn from that report, with some reorganization of the material. Arthur D. Little, Inc., has not conducted further analyses or literature searches.

The methodology adopted by JRB involved acquiring the most recent industry data on production and consumption, by process. If release rates had been published, they were used to predict releases for each process. In tables and figures reporting release data and estimates, the term "releases to land" includes material applied to the soil as well as material in landfills. All data are 1978 figures unless otherwise stated.

The materials balance of benzene is somewhat unusual in that nearly equivalent quantities originate from "indirect" and "direct" sources. Direct sources are those deliberately involved in the production and use of benzene, while indirect sources are not. Forty-four petroleum refineries produce benzene as part of their normal operations. Ten coke manufacturing plants and an unspecified number of petroleum plants that refine light oils from coking operations also engage in the production of benzene. Direct sources also include imports and inventory withdrawals.

Present in varying concentrations, benzene is a constituent of coal and crude oil; therefore some refineries and coke plants are direct sources of benzene. The remaining plants, though indirect sources, may also be sources because of their production of benzene in the form of products and process emissions. Thus, petroleum refining, specifically because of the production of gasoline, is an indirect source of benzene, as well as coal coking operations without benzene recovery, oil spills, and mining and resource processing.

The materials balance for benzene is summarized in Table 3-1, which shows the amounts of benzene for direct and indirect sources including the uses, releases, destruction, and amounts of benzene unaccounted for. JRB notes that the subtotal for production etc. available for the direct use of benzene is not equivalent to the subtotal for direct use. This discrepancy occurred because separate data bases were used for the two sets of calculations.

TABLE 3-1. MATERIALS BALANCE FOR BENZENE, 1978

Direct Sources	kkg Amount	kkg Releases	Uses	kkg Amount	kkg Releases	kkg Destruction[a]	kkg Unaccounted
Refining Production from Crude	4,709,900	3,900	Chemical Feedstock	5,251,000	10,916 10,865[b]	5,107,300	121,919
Refinery Production from Light Oil, etc.	65,400	?					
Coke Plant Production	178,786	786	Solvent Use	9,600	2,510	6,590	0
Inventory Withdrawals	272,000	0	Exports	151,000	17	150,983	0
Imports	225,000	26	Transport & Storage[d]	-	7,272	0	-7,272
Subtotal	5,451,086	4,712		5,411,600	31,580	5,264,873	114,647
Indirect Sources							
Gasoline Refining	1,400,000	20,230[c]	Gasoline Consumption	4,665,770	130,059	4,481,670	0
Gasoline Imports	3,288,000		Transport & Storage	-	21,000		0
Other Fuels	959,410		Fuel Use	959,410	40,213[e]	919,197[e]	
Coal Coking	?	1,872					
Contaminated Solvents	22,600		Other Solvents	22,600	7,100[f]	15,520[f]	0
Oil Spills	30	30					
Resource Mining/Processing	148	148					
TOTALS	11,121,274	26,992		11,059,380	219,087	10,681,240	114,647

Balance: Sources - Releases ≟ Uses ≟ Releases + Carryover + Destroyed + Unaccounted for

11,094,282 ≟ 11,059,380 ≟ 11,025,839

Discrepancy 34,902 + 33,541 = 68,443 = 0.6% of total sources.

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[a] Amount destroyed includes amounts transformed, transferred or otherwise chemically altered.

[b] Carryover into products; not included in column total.

[c] Includes 2,000 kkg destroyed during refining (not described as a release elsewhere).

[d] These releases occur from all sources and users, save gasoline and fuels, and required a negative entry to avoid double accounting.

[e] Estimate based on percent released and percent destroyed for gasoline.

[f] Estimate based on percent released and percent destroyed for solvent use.

Source: JRB (1980).

3.2 PRODUCTION OF BENZENE

Benzene is produced commercially from three raw material sources: petroleum by various methods; coal during coke production; and condensates from gas wells. Although originally coal was the commerical source of benzene, petroleum is the primary source of benzene today (Kirk-Othmer 1976). According to the USITC (1976) 178,000 kkg of benzene were obtained directly from coal and 65,400 kkg from light oils produced by coal coking, while 4,710,000 kkg were produced by refining petroleum.

3.2.1 Direct Production from Oil

Benzene is a component of crude oil in concentrations ranging from 0.001 to 0.4% (Versar, Inc. 1977). Recovery of benzene is not cost effective unless a catalytic or thermal reaction increases its concentration.

The four basic commercial methods used to increase the benzene concentration in petroleum and then isolate it are:

- Catalytic reformation of the naphtha petroleum fraction;
- Dealkylation of toluene;
- Disproportionation of toluene, and
- Isolation from pyrolysis gasoline as a by-product of ethylene manufacture.

The names, locations, production amounts and processes of the companies that produce benzene from petroleum are presented in Table 3-2. They are shown on a U.S. map (see Figure 3-1), which also shows the sites of benzene production from petroleum and coal.

Total generalized releases of benzene from the four petroleum production processes based on process efficiencies were estimated to be 3900 kkg for 1978. Table 3-3 lists releases and production from each process. A wide range exists between estimates presented by JRB based on available release factors: 30-50,000 kkg/yr. JRB estimated overall benzene releases from petroleum production of benzene to be closer to the 3900 kkg/yr figure than to the more extreme values. JRB based this conclusion on the judgment that for economic reasons, all feasible control methods would be implemented to prevent undue loss of a product. Several industrial contacts confirmed this evaluation.

No air release factors for petroleum refinery production of benzene were found.

The solid wastes generated from benzene production include the following types: solid, liquid-solid slurries, and sludges. The components of the solid wastes that appear to come directly from the benzene syn-

TABLE 3-2. PRODUCERS AND PRODUCTION OF BENZENE, 1975-79

Company	Location	Estimated Production[a] (kkg) 1979	1978	1977	1976	1975	Production Processes[b] and Use
Allied Chemical	Winnie, TX				8,520	5,680	
Amerada Hess Corp.	St. Croix, Virgin Islands	162,000	146,000	159,000	71,000	47,400	CR
American Petrofina, Inc.	Port Arthur, TX				42,600	28,400	C
(Cosden Oil & Chemical Co.)	Big Spring, TX	144,000	130,000	142,000	128,000	85,300	CR, TD
Ashland Oil, Inc.	Ashland, KY	159,000	144,000	156,000	142,000	95,000	CR, TD, LO
	North Tonawanda, NY	57,200	51,700	56,200	42,600	28,400	CR, LO
Atlantic Richfield Co.	Houston, TX	139,000	126,000	103,000	125,000	83,400	CR, TP
	Wilmington, CA	29,800	27,000	29,300	34,100	22,700	CR
	Channelview, TX	162,000	146,000	78,200			PG
Charter International Oil Co.	Houston, TX	12,400	11,200	12,200	14,200	9,470	CR
Cities Service Co., Inc.	Lake Charles, LA	62,100	56,200	61,100	71,000	47,400	CR
Coastal States Gas Prod. Co.	Corpus Christi, TX	174,000	157,000	171,000	199,000	133,000	25% C, CR, TD
Commonwealth Oil Refining Co. (Commonwealth Petrochemicals)	Penuelas, Puerto Rico	460,000	416,000	452,000	525,000	350,000	PC, CR, TD, PG
Crown Central Petroleum Corp.	Pasadena, TX	57,200	51,700	56,200	65,300	43,600	CR, TD
Dow Chemical Co.	Bay City, MI	74,600	67,500	73,300	85,200	56,900	C, TD, PG, LO
	Freeport, TX	124,000	112,000	122,000	142,000	94,700	C, TD, PG
	Plaquemine, LA	149,000					
Eastman-Kodak Co. (Texas Eastman Div.)	Longview, TX	?	?	?	?	?	
Exxon Corp.	Baton Rouge, LA	174,000	157,000	171,000	185,000	123,000	CR, PG
	Baytown, TX	149,000	135,000	147,000	176,000	117,000	67% C, CR
Getty Oil	El Dorado, KS	32,300	29,200	31,800	37,000	24,600	C, CR
Gulf Oil Corporation	Alliance, LA	167,000	151,000	164,000	199,000	133,000	C, CR, TD
	Philadelphia, PA	92,000	83,200	90,400	93,700	62,500	C, CR, TD
	Port Arthur, TX	186,000	169,000	97,700	108,000	72,000	C, CR, PG
Kerr-McGee Corp. (Southwestern Oil & Ref. Co.)	Corpus Christi, TX	39,800	36,000				
Marathon Oil Co.	Texas City, TX	17,400	15,700	17,100	17,000	11,400	C, CR
Mobil Oil Corp.	Beaumont, TX	149,000	135,000	147,000	170,000	114,000	CR, PG
Monsanto Co.	Chocolate Bayou, TX	211,000	191,000	208,000	213,000	142,000	C, CR, TD, PG
Pennzoil United, Inc. (Atlas Processing)	Shreveport, LA	87,000	78,700	36,600	42,600	28,400	PG
Phillips Petrol. Co.	Sweeny, TX	24,900	22,500	24,400	62,500	41,700	C, CR
	Guayama, Puerto Rico	273,000	247,000	269,000	312,000	208,000	PC, CR, TD
Quintana-Howell	Corpus Christi, TX	224,000	202,000	17,100			CR

Sources: Arthur D. Little, Inc. (1977), SRI (1977), Versar, Inc. (1979), Neufeld _et al._ (1978).

TABLE 3-2. PRODUCERS AND PRODUCTION OF BENZENE, 1975-79 (Continued)

Company	Location	Estimated Production[c] (kkg) 1979	1978	1977	1976	1975	Production Processes[b] and Use
Shell Oil Co.	Deer Park, TX	298,000	270,000	220,000	213,000	142,000	C, CR, PG
	Odessa, TX	29,800	27,000	29,300	17,000	11,400	CR, TD
	Wood River, IL	112,000	101,000	110,000	114,000	76,000	CR, LO
Standard Oil Co. of Calif.	El Segundo, CA	57,200	51,700	56,200	65,300	43,600	C, CR
Standard Oil Co. (Ind.)(AMOCO)	Texas City, TX	211,000	191,000	208,000		161,000	C, CR
Standard Oil Co. (Ohio) (B.P. Oil Co.)	Marcus Hook, PA				22,700	15,200	
Sun Oil Co.	Marcus Hook, PA	72,100	65,200	70,800	42,600	28,400	CR, TP
	Corpus Christi, TX	94,500	85,400	92,800	99,400	66,300	C, CR, TD
	Tulsa, OK	59,700	54,000	58,600	63,200	45,500	CR, TP
	Toledo, OH	184,000	166,000	120,000			CR, TD
Tenneco, Inc.	Chalmette, LA	24,900	22,500	24,400	28,400	18,900	CR, LO
Texaco, Inc.	Port Arthur, TX	112,000	101,000	110,000	128,000	85,300	73% C, CR
	Westville, NJ	87,000	78,700	85,500	99,400	66,300	PC, CR
Union Carbide Corp.	Taft, LA	174,000	157,000	171,000	199,000	133,000	C, PG
Union Oil Co. of Calif.	Lemont, IL	42,300	38,200	41,500	54,000	36,000	CR, LO
Union Oil-American Petrofina	Beaumont, TX	54,700	49,500	53,700	54,000	36,000	50% C, TD
Union Pacific Corp. (Champlin Petroleum Co.)	Corpus Christi, TX	24,900	22,500	24,400	28,400	18,900	C, CR
TOTAL USITC PRODUCTION		5,430,000[c]	4,780,000	4,570,000	4,540,000	3,190,000	

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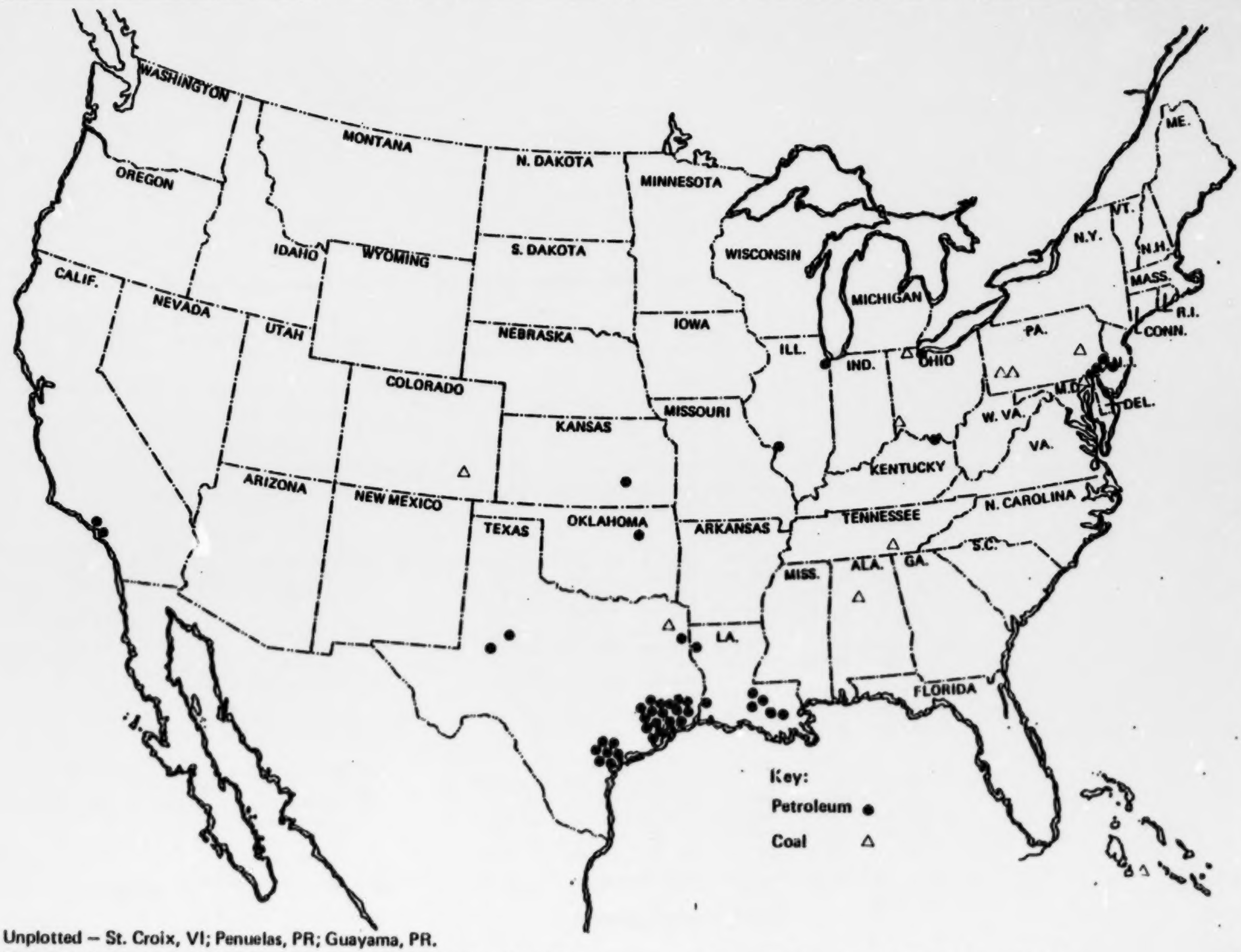
[a] Derived from plant capacities and USITC production totals.

[b] Key: C, captive use; PC, partially captive; CR, catalytic reformation; TD, toluene alkylation; TP, toluene disporportionation; PG, pyrolysis gasoline; LO, light oil; CS, gaswell condensate.

[c] Estimated by extrapolating USITC data for the months January through July 1979.


WASHINGTON
OREGON
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NEVADA
IDAHO
MONTANA
WYOMING
UTAH
COLORADO
ARIZONA
NEW MEXICO
N. DAKOTA
S. DAKOTA
NEBRASKA
KANSAS
OKLAHOMA
TEXAS
MINNESOTA
IOWA
MISSOURI
ARKANSAS
LA.
WISCONSIN
ILL.
MICHIGAN
IND.
OHIO
KENTUCKY
TENNESSEE
MISS.
ALA.
GA.
FLORIDA
S.C.
N. CAROLINA
VA.
W. VA.
PA.
N.Y.
VT.
N.H.
ME.
MASS.
R.I.
CONN.
N.J.
DEL.
MD.
Key:
Petroleum ●
Coal △

Unplotted – St. Croix, VI; Penuelas, PR; Guayama, PR.

Sources: Arthur D. Little, Inc. (1977), SRI (1978), Versar, Inc. (1977), Neufeld *et al.* (1978).

FIGURE 3–1 PRODUCERS OF BENZENE FROM PETROLEUM AND COAL, 1978

TABLE 3-3. BENZENE RELEASES FROM DIRECT PETROLEUM PRODUCTION, 1978

	Benzene Produced (kkg)	Estimated General[a] Releases
Catalytic reformation	2,360,000	2,360
Toluene dealkylation	1,300,000	1,300
Toluene disproportionation	121,000	60
Pyrolysis gasoline	925,000	180
Total	4,706,000	3,900
Total Production based on USITC data	4,775,300	

---

[a] Release factors are given by process for total releases. In the text, specific release factors are given only for benzene production from petroleum as a whole.

thesis processes are acid and alkali sludges (Saxton and Narkus-Kramer 1975). The quantity of solid waste generated from benzene production was calculated using data of Saxton and Narkus-Kramer, who calculated the amount of solid waste generated from benzene production in 1972. These figures include benzene production for all processes. Benzene releases in solid wastes from petroleum-based production were 141 kkg. It was assumed that these wastes would be handled as are refining wastes, which are landfilled. Because benzene represents only 1% of the total amount of waste generated, it must be realized that the actual quantities (volumes) of the wastes described above are 100 times larger.

Air releases were estimated by the difference between the total releases (3900 kkg) and those attributed to land (141 kkg) and water (620 kkg). Thus, air releases would be about 3140 kkg.

Releases from refining of gas well condensates were not estimated because of the lack of release factors specific to the process.

3.2.2 Direct Production from Coal

Benzene is obtained from coal by extraction from the light oil formed during coking. Crude light oil consists of 55-70% benzene by volume (Arthur D. Little, Inc. 1977). The yield of light oil from coke ovens producing blast furnace coke is 11.4-15.1 l/kkg of coal carbonized (PEDCo 1977). The light oil is refined by various processes that result in separation into benzene, toluene, xylene, and residue fractions.

The treatment of coal tar may also be used to obtain light oil. The tar can be distilled to yield a light oil fraction, which is usually combined with the light oil from coal gas before it is refined to produce benzene (PEDCo 1977). Light oil is either refined on site or it is sold. Several petroleum producers refine this coal-derived light oil (SRI 1978, Arthur D. Little, Inc. 1977).

In 1978, 254,000 kkg of coal-derived benzene were produced (USITC 1978). This represented 4% of total benzene production. Ten plants refined their own light oil and produced 178,000 kkg of benzene in 1978. An unspecified number of coke plants sell their light oil to refineries for benzene extraction. This quantity, 65,400 kkg, was previously accounted for in the section covering petroleum refinery production of benzene with respect to emissions and total production. Table 3-4 is a list of producers who derive benzene from coking operation oils or those who generate the light oil and sell it to refineries.

Benzene releases from coal coking operations, refining of coal-derived light oil operations (for which no release factors were found) are mostly gaseous and some liquid. The amount of benzene released

TABLE 3-4. PRODUCERS OF BENZENE FROM COKE

Company	Location	Production in 1978 (kkg)
Armco Steel Corp.	Middletown, OH	5,700
Bethlehem Steel Corp.	Bethlehem, PA	7,700
	Lackawanna, NY	0
	Sparrows Point, MD	29,000
Mead Corporation	Chattanooga, TN	0
	Woodward, AL	3,800
C.F. & I. Steel Corp.	Pueblo, CO	5,700
Interlake, Inc.	Toledo, OH	1,900
Jones & Laughlin Steel Corp. (LTV Corp)	Aliquippa, PA	19,000
Northwest Industries, Inc. (Lone Star Steel Corp)	Lone Star, TX	1,900
U.S. Steel Corp.	Clairton, PA	96,000
	Geneva, UT	7,700
Total Production		178,062

Source: Adapted from JRB (1980).

to the air during coking operations was estimated using three factors from the literature. When the factor developed by Walker (1976) was used to estimate the release of 59,000 kkg benzene, it was assumed that coking production was at full capacity, requiring consumption of 88,000,000 kkg of coal (derived from Table C-1 in Mara and Lee 1978), and that the yield of coke from coal is 68.4%. The value 88,000,000 kkg of coal was used in calculations with the other release factors. With the PEDCo (1977) factor, benzene releases to air from all coking operations were calculated as 6900 kkg based on U.S. EPA (1977) data for atmospheric emissions. JRB judged the Mara and Lee factor to be most accurate and estimated releases of 2640 kkg.

Based on data contained in the JRB report, a maximum air release was estimated for coking operations that derive benzene from the light oils produced during coking. This calculation does not include air releases, which may occur during refining of light oil for benzene, nor releases attributed to coking operations that do not produce benzene. The ten coking facilities producing benzene have the capacity for 25,600,000 tons of coal. Using the emission factor of Mara and Lee (1978), for every ton of coal coked, the potential release of benzene to the air if all ten facilities operate at full capacity is:

$$2.56 \times 10^{7} \text{ kkg coal} \times 3 \times 10^{5} \frac{\text{kkg benzene}}{\text{kkg coal}} = 768 \text{ kkg.}$$

In the absence of emission factors for the refining process of either light oil or coal tar refining for light oil, 768 kkg will be used as a minimum air release for coal-derived benzene production, even though the assumption of full capacity is incorrect. Versar, Inc. (1980) estimates benzene discharges to water for coking operations to be <10 kkg. JRB also accepted this figure.

Possible sources of release during production of benzene from light oils are predominantly liquids: shock liquors, aqueous effluents, oil, wash oil, light oil, etc., and solids in the forms of tars.

No release factors were given for air or water for benzene extraction from coal tars. Land releases, however, were estimated to be 8 kkg.

3.3 IMPORTS AND EXPORTS OF BENZENE

Benzene imports amounted to 225,000 kkg in 1978. The estimated releases attributed to importing were 13 kkg to air and 13 kkg to water. Releases due to imports are the result of unloading operations and transport to points of consumption. Using release factors developed by PEDCo (1977) and assuming 95% emission control at dockside and a 50/50 split between air emissions and water discharges, the estimated benzene release is 13 kkg to each of the two media. No solid wastes are generated during importation .

Exports accounted for 151,000 kkg of benzene in 1978. Estimated releases due to exportation (dockside loading activities) were 15 kkg to land and 2 kkg to water. The lack of air emissions from imports, and land releases for exports reflect the different modes of transport involved in these two transfers. Although the same transportation release factor is used for exports and imports, JRB believes that 90% of transport releases are to air, because most losses will be overland spills, in which case, most of the spill would evaporate. This is a fate estimate, therefore, the 15 kkg is reported here as releases to land. The PEDCo release factor for export dockside loading is much smaller (10^{-4}) than the dockside unloading factor used for imports. Thus, they are eclipsed by transportation losses.

3.4 INDIRECT SOURCES OF BENZENE

Benzene may be released from the following indirect sources:

- Coal coking,
- Petroleum refining for gasoline,
- Use of products (mostly solvents) contaminated with benzene,
- Natural gas well condensates,
- Resource mining and processing,
- Oil well drilling,
- Oil spills, and
- Combustion.

3.4.1 Coal Coking

Coal coking operations do not involve the collection of light oils that are refined for benzene. Of the 88,000,000 kkg of coal coked in 1978, 62,400,000 kkg were coked at facilities without benzene production. Based on the release factor of Mara and Lee (1978) used in Section 3.2.1.2, the amount of benzene released to the air during coking at these plants is:

$$62.4 \times 10^6 \text{ kkg coal} \times 3 \times 10^{-5} \frac{\text{kkg benzene}}{\text{kkg coal}} = 1872 \text{ kkg.}$$

This amount in addition to the 768 kkg attributed to coking operations at plants that do produce benzene equals 2640 kkg. This value corresponds to total coking industry benzene releases to air, which assumes that all plants condense their light oils and do not release them to the air in the gaseous state.

Release factors were not available for either land or water releases from coking operations.

3.4.2 Petroleum Refining for Gasoline

It is estimated that crude oil contains an average of 0.2% benzene (Walker 1976). Therefore, petroleum refining operations are expected to be a source of benzene releases. The amount of crude refined in the United States was 5×10^9 bbls (2.1×10^{11} gal) in 1978. This amount contains 4.2×10^8 gal of benzene or 1.4×10^6 kkg (one liter of benzene weighs 0.878 kg).

The literature revealed several release factors for benzene of air (Mara and Lee 1978, PEDCo 1971, Versar, Inc. 1977). Of these, JRB has used the data based on Mara and Lee's factor to provide a maximum. Air releases are estimated to be 20,000 kkg/yr.

One factor for releases to water from petroleum refining was calculated from data presented by Versar, Inc. (1977). Sampling data for six refineries were collected by Versar for the Effluent Guidelines Division of EPA. Of these, one had a benzene concentration of 7 μg/l in its effluent, while no benzene was detected in effluents of the other five. JRB assumed full-capacity production and direct discharge of all effluents, combined with a release factor derived from the PEDCo figure to estimate water releases of 1 kkg for the industry in 1978.

The amount of benzene in solid waste resulting from petroleum refining was between 71 and 230 kkg (Table 3-5) for 1978. In 1976, the American Petroleum Institute's survey of the industry revealed a waste amount of 357,000 kkg for the year. In the United States, 4897×10^6 bbl (6.666×10^8) of crude were processed; 39% of which was not domestic oil. Using these two figures, a waste generation factor of 0.54 kg/kkg was derived. Using the conversion factors of JRB (75% recovery; 16% of waste is oil; benzene content averages 0.5%), the amount of benzene in refinery wastes was 71.4 kkg.

The waste generation factor used by JRB results in yields of more waste than crude (1.64 kkg/kkg). Assuming that this factor is off by 10^{-3}, the amount of benzene in refinery wastes was recalculated to be 229.8 kkg.

The total amount of benzene released to the environment during refining operations is 20,230 kkg in 1978. Of the initial amount available in crude (1,400,000 kkg), 1,379,770 remains in the refined gasoline product.

3.4.3 Use of Products Contaminated with Benzene

The three solvents co-produced with benzene are toluene, xylene, and hexane. Their estimated benzene contamination is 0.001-0.04% by weight. One order of magnitude estimate of the quantity of benzene

TABLE 3-5. PETROLEUM INDUSTRY DISPOSAL OF SOLID WASTES CONTAINING BENZENE

Disposal Method[a]	Refining (%)[a]	Solid Wastes (kkg)	Benzene Disposed (kkg)
Landfilling	51.1	5.88×10^8	116
Landspreading	8.4	0.97×10^8	19
Lagooning	39.7	4.57×10^8	91
Incineration	0.8	0.09×10^8	2
TOTAL			228

---

[a] Adapted from Jacobs (1978) by JRB.

in these products is given as 22,600 kkg (see Table 3-6). Actual releases are unknown. However, if the proportions of destroyed and released benzene developed for pure benzene solvent are applied to contaminated solvents (see Section 3.5.2.1), it can be estimated that 15,500 kkg (68.6%) are destroyed and 7100 kkg (31.4%) are released to the environment.

Other petroleum products that may contain from trace to 3% benzene (by volume) are solvent naphthas (aromatic petroleum, Stoddard, VM&P, etc.), coke-oven tar, and lubricating oils. Production figures were not available for these products. Because of their nature, any contaminating benzene could come in direct contact with consumers. Losses would be evaporative, aqueous (washing), and solid (municipal wastes).

3.4.4 Natural Gas Well Condensates

Benzene is a component of gas well condensates. Atlas Processing, a subsidiary of Pennzoil, was reported to produce small quantities of benzene (SRI 1978). The company's benzene-producing wells are located in the East Texas gas fields. JRB postulated that other gas wells in the region also contain benzene. However, attempts were unsuccessful in obtaining information on the fate of these condensates or well-head release rates. Further study is needed to determine the number of gas wells that have condensates containing benzene, the quantity of benzene contained in the condensates, and the types and quantities of releases to the environment. These sources are of potential significance.

3.4.5 Resource Mining and Processing Operations

The mining and processing of mineral, timber, and fiber resources produced some benzene releases to water. Table 3-7 shows estimates of benzene releases to water totaling 148 kkg from these resources. No additional information or process descriptions could be obtained from Versar, Inc. (1977) who originated these estimates.

3.4.6 Benzene Releases from Oil Well Drilling

The drilling of oil wells produces environmental releases of benzene from drilling fluids, muds, and uncontrolled flow of crude oil above or below the surface. The quantity of benzene releases depends on the percentage of benzene in the crude and the extent of uncontrolled crude flow, which may contain drilling muds and fluid. No information was obtained on the quantity of benzene involved in this potential source of environmental release. JRB estimates that oil drilling sites are a potentially significant source of benzene releases.

TABLE 3-6. BENZENE IN CONTAMINATED SOLVENTS

Solvent	Quality Produced in 1978[a] (kkg)	Estimated Benzene Contamination (% by Weight)[b]	Amount of Benzene as Contaminant[c] (kkg)
Toluene	390,000[d]	0.04	15,600
Xylene	2,915,000	0.001	3,000
Hexane	200,000	0.02	4,000

---

[a] USITC (1978).

[b] Arthur D. Little, Inc. (1977).

[c] Considered to be order of magnitude estimates.

[d] SRI (1979).

TABLE 3-7. GROSS ANNUAL DISCHARGES OF BENZENE TO WATER FROM RESOURCE MINING AND PROCESSING, 1976

Process	Estimated Discharge of Benzene to Water (kkg/yr)
Nonferrous metals manufacturing (Al, Cu)	2.85
Ore mining (Pb, Zn)	1.1
Wood processing	0.4
Coal mining	141.1
Textile industry (SIC subcategories 40 and 60)	2.51
Total	148

Source: Versar, Inc. (1977).

3.4.7 Benzene Releases from Oil Spills

The environmental release of benzene from oil spills depends on the quantity of benzene in the crude oil, the size of the spill, the frequency of spills, and the location of the spill (surface of subsurface).

Walker (1976) estimated benzene releases to the oceans from oil spills of all types to be 10,500 kkg/yr.

Versar, Inc. (1977) estimated the gross annual discharge of benzene to U.S. waters from U.S. Coast Guard information on crude oil spills in 1976 to be 30 kkg.

3.4.8 Combustion of Petroleum-based Fuels

3.4.8.1 Benzene in Gasoline

Benzene is an important constituent of gasoline. In 1978, 1.13623×10^{11} gal (3.8×10^{8} kkg) of motor gasoline were supplied for domestic use (U.S. DOE 1979). This figure includes U.S. production, releases from inventories, and imports less exports. In 1978, JRB estimated the total amount of benzene in gasoline as 4.4×10^{6} kkg, based on the average benzene concentration (1.17%) in gasoline. The amount of benzene used annually in motor fuels that originates abroad or is withdrawn from inventories is 3,288,000 kkg, while 1,152,000 kkg originated within the United States in 1978.

Benzene is present at low concentration in crude oil. In the catalytic reformation process, the benzene content of the crude is increased when longer-chain molecules are broken down. This reformate, containing the aromatics (including benzene), makes up about 20% of the "pool" of materials in gasoline. During refining, BTX (a mixture) of benzene, toluene, and xylene) is often separated from reformate. BTX may be blended back into the gasoline pool to increase the concentration of benzene in the gasoline in order to raise its octane rating, (USITC 1978). Thus, if benzene is used as an octane-raising additive, it is added to the gasoline pool as a component of BTX and not as pure benzene. In this situation, the benzene would not be counted in reports of total benzene consumption because it is not separated from the BTX. Therefore, gasoline production, in addition to benzene production, significantly contributes to the amount of benzene released to the environment. A materials balance for the benzene is gasoline, therefore, may be considered independently from a materials balance for benzene in general.

Because the petroleum refineries that produce gasoline also engage in other processes, any release of benzene from gasoline production would be included in releases from petroleum refineries. These releases, which total 248,000 kkg, were detailed previously (see Section 3.4.2) and will not be described further.

A flow diagram of gasoline from production center to its ultimate combustion in a motor vehicle engine is represented in Figure 3-2. The distribution system, which transports gasoline from the petroleum refineries to the consumer with intermediate storage stops, is a source of atmospheric benzene (21,100 kkg). Gasoline is shipped from refinery storage areas to bulk terminals (regional distribution centers) by ship, barge, railcar, and pipeline. Then it is transported from the terminal by tank truck to service stations and commercial and rural users, either directly or via bulk plants (local distribution centers) (Burklin et al. 1975, PEDCo 1977, Mara and Lee 1978). Benzene releases to air associated with particular segments of this flow are the maximum estimates given by JRB in each case (see Figure 3-2).

The benzene concentration in gasoline depends on several factors, including the source of the crude oil from which the gasoline was made, the location of the crude oil source and the refiner, the grade of gasoline, refinery operations, and the seasonal blends produced by each refinery (PEDCo 1977).

The lowest reported benzene concentration in the surveys referred to above was 0.25% by volume (premium, summer, district 2). The highest benzene level was 3.91% (unleaded, winter, district 4). National averages from the reports (in percent by volume) were as follows:

	Unleaded	Regular	Premium
Summer	1.20	1.19	1.10
Winter	1.26	1.12	1.15

Because the differences in benzene concentration between fuels of different grades and seasonal blends were smaller than the variation within each blend or grade, JRB chose the average of the above values, 1.17%, to represent the benzene concentration in all gasolines for calculating releases and total amount of benzene in gasoline. Therefore, the total amount of benzene in gasoline was 4.4×10^6 kkg in 1978. Of this amount, 21,000 kkg are the estimated air releases due to evaporation, venting, etc., during storage, transfer and transportation operations, which convey the gasoline to the final consumer. Emissions attributed to vehicular use are from engine exhausts, and evaporation from the carburetor, etc. JRB calculated these releases as totaling between 53,894 and 165,521 kkg. At maximum releases, 52% is attributed to autos, 12% to motorcycles, and 36% to trucks and buses. Auto exhaust releases were recalculated on the basis of in-house data (unpublished), which indicated that the emission rates used by JRB were high and possibly the results from outdated, less accurate analyses than are possible today. The new figures result in a total air release between 45,989 and 130,059 kkg. At maximum releases, 39% is attributed to autos, 14% to motorcycles, and 47% to trucks and buses. These calculations are given in Appendix A.

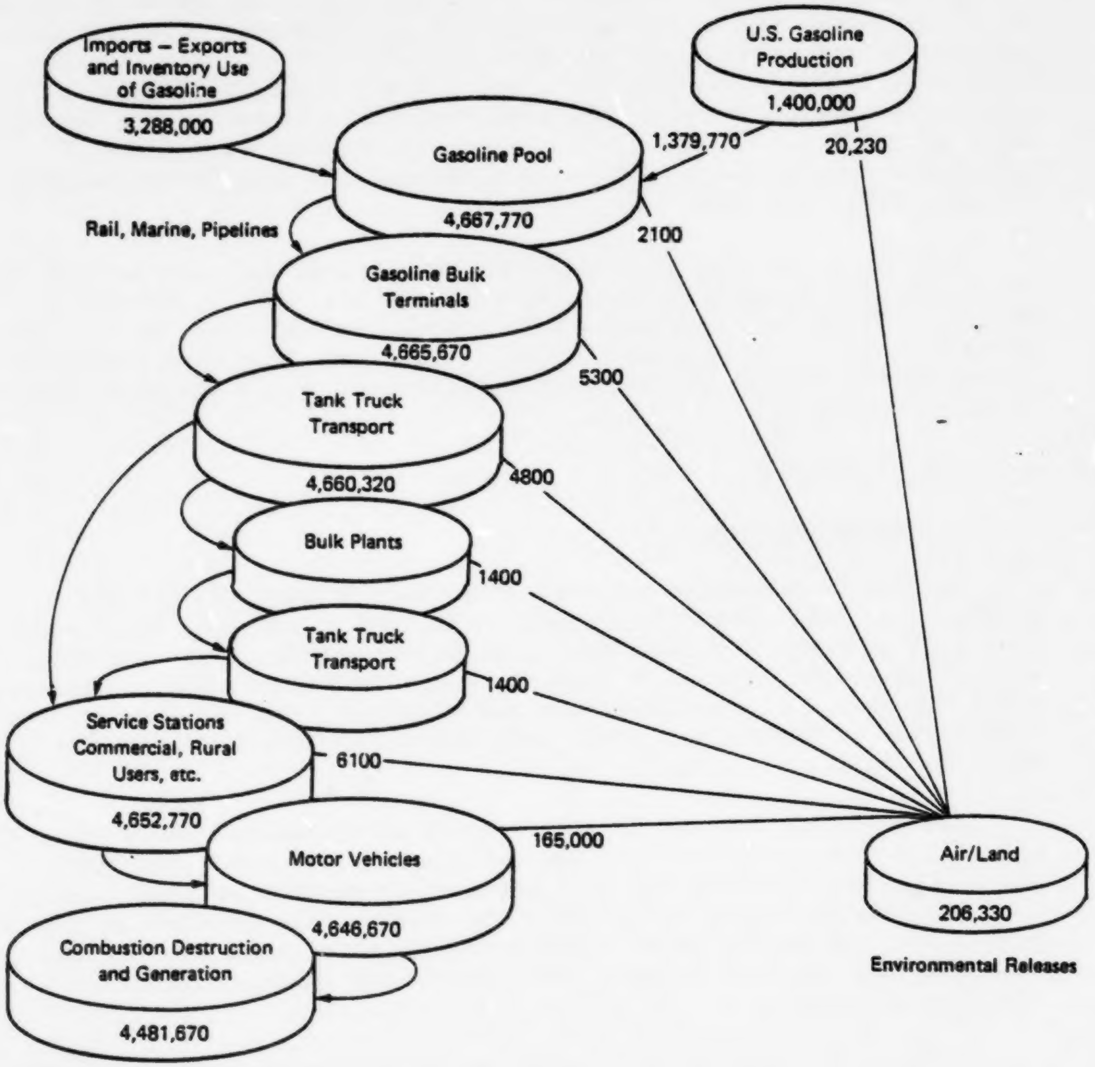
Imports – Exports and Inventory Use of Gasoline
3,288,000
U.S. Gasoline Production
1,400,000
1,379,770
20,230
Gasoline Pool
4,667,770
Rail, Marine, Pipelines
2100
Gasoline Bulk Terminals
4,665,670
5300
Tank Truck Transport
4,660,320
4800
Bulk Plants
1400
Tank Truck Transport
1400
Service Stations Commercial, Rural Users, etc.
4,652,770
6100
Motor Vehicles
4,646,670
165,000
Air/Land
206,330
Environmental Releases
Combustion Destruction and Generation
4,481,670

FIGURE 3–2 MATERIALS BALANCE FOR BENZENE IN GASOLINE IN KKG

3.4.8.2 Benzene in Other Petroleum-based Fuels

The benzene concentrations of eight fuels were estimated by Arthur D. Little, Inc., (1977) and are presented in Table 3-8. The estimated benzene content calculated from these concentrations is 959,410 kkg. These calculations indicate that a significant quantity of benzene is present in aviation turbine fuel, which consists of naptha and kerosene types of jet fuel. Because of the magnitude of this estimate, JRB recommends further investigation to determine the quantity of benzene released from this fuel.

For the purpose of this materials balance, it has been assumed that the total 1978 production of these fuels was used and that a similar fraction (95.8%) of the benzene therein would be destroyed during combustion as it is in gasoline. Thus, 919,197 kkg would have been destroyed during the use of nongasoline fuels, and 40,213 kkg would be released to the air.

3.5 USE OF BENZENE

3.5.1 Consumptive Use

Benzene is predominantly used as a starting material for the synthesis of other organic compounds. In 1978, 5,230,000 kkg of benzene were consumed by production of these eight compounds: ethylbenzene, cumene, cyclohexane, nitrobenzene, chlorobenzene, chlorobenzenes, alkyl benzenes, maleic anhydride, and biphenyl. The eight major direct derivatives of benzene and their contributors to total benzene consumption are listed in Table 3-9.

The materials balance for benzene use as a chemical feedstock, showing carryover, destruction and environmental releases totaling 10,916 kkg is given in Table 3-10 for each production process. JRB used several references to estimate the release factors shown in the footnotes. The values given represent JRB's best judgment of actual releases, when several release factor estimates were available for a specific process.

Although the release rates were not available to estimate benzene in solid wastes from consumptive use processes, its presence in these wastes cannot be overlooked during fate and exposure analyses.

The possibility of product contamination by benzene was examined for the eight products and their major derivatives. In all cases, test estimates indicated that benzene carryover was <1% and usually <0.1%. The maximum carryover was estimated for each process and the total amount was 10,866 kkg, or 0.2% of benzene use as feedstock. To complete the materials balance for these processes, it was necessary to base calculations on theoretical process efficiencies in order to obtain quantity of benzene chemically transformed (or destroyed) during the eight processes.

TABLE 3-8. ESTIMATED BENZENE CONTENT OF FUELS

Benzene-Containing Fuels[a]	Fuel Produced in 1978[b] (gallons)	Estimated Benzene Concentration (% by Volume)	Benzene Produced as a Component of Fuel (kkg)
Aviation Gasolines	5.85×10^8	0.4 - 3[d]	38,000
Farm Tractor Fuels	?	0 - trace[e]	?
Diesel Fuel Oils	5.26×10^{10}	0 - trace[e]	200
Aviation Turbine Fuels[c]	1.62×10^7	0 - 3[d]	921,000
Gas Turbine Fuel Oils	?	0 - trace[e]	?
Liquefied Petroleum Gases	1.5×10^7	0	0
Fuel Oils	4.63×10^7	0 - trace[e]	200
Kerosene	2.7×10^6	0 - trace[e]	10
Total Estimated Benzene Content			959,410

---

[a] Arthur D. Little, Inc. (1977).

[b] U.S. DOE 1979, the data were connected from barrels to gallons using the conversion factor of 42 gallons/barrel.

[c] Jet fuel is the total of naphtha and kerosene types.

[d] An arithmatic average was used in the calculation of benzene concentration.

[e] Arthur D. Little, Inc. (1977) gave no indication of what "trace" meant. JRB, Inc. assumed that trace is 1 part per million.

TABLE 3-9. SUMMARY OF CONSUMPTIVE USES OF BENZENE, 1978

Product	Secondary Products or Uses	Production[a] (kkg)	Benzene Required[b] (kkg)	Consumptive Use (% of Total)
Ethylbenzene	Styrene; polystyrene	3,803,000	2,810,000	53.5
Cumene	Phenol	1,533,000	1,030,000	19.6
Cyclohexane	Cyclohexanone; nylon 66	1,057,000[d]	836,000	15.9
Nitrobenzene[c]	Aniline	261,000	170,000	3.2
Chlorobenzenes	Chemical intermediates	172,000	134,000	2.6
Alkylbenzenes	Detergents	330,000[e]	132,000	2.5
Maleic anhydride	Chemical intermediates	155,000[f]	132,000	2.5
Biphenyl	PCBs; dyes	29,000[g]	7,000	0.1
TOTALS		7,340,000	5,251,000	99.9

[a] USITC figures except where noted.

[b] Conversion factors from Neufeld et al. (1978).

[c] Includes nitrobenzene destined for aniline synthesis (96%) plus nonaniline usage (4%).

[d] 85% was derived from hydrogenation of benzene (Blackford 1977).

[e] Derived from USITC (1978) production figure for linear to (239,000 kkg) using the 1978 ratio for capacities of linear to branched alkylbenzenes of 2.63 (Bradley 1979, Chemical Marketing Reporter 1979).

[f] 84% was derived from oxidation of benzene (Gerry et al. 1979).

[g] 30% was derived from thermal dehydrogenation of benzene.

TABLE 3-10. MATERIALS BALANCE FOR BENZENE IN CHEMICAL FEEDSTOCKS

					Benzene Concentration (kkg)				
		Estimated Releases[a] to							
Product Synthesized	% Yield	Air	Water	Land	Total Releases	Amount Used	Amount Destroyed[d]	Maximum Carryover	Amount Unaccounted
Ethylbenzene	99	3,900	120	NK[b]	4,020	2,810,000	2,782,000	160	23,820
Cumene	96	2,000	40	NK	2,040	1,030,000	988,830	15	39,115
Cyclohexane	99	290	0	NK	290	836,000	827,640	5,300	2,770
Nitrobenzene	97	340	16	NK	356	170,000	164,900	4,730	14
Maleic Anhydride	70	3,600	8	0	3,608	132,000	92,400	160	35,832
Chlorobenzene	85	340	16	NK	356	134,000	113,915	170	19,559
Alkybenzenes	99[c]	170	35	NK	205	132,000	130,685	330	780
Biphenyl	99[c]	41	NK	NK	41	7,000	6,930	0.09	28.9
TOTALS		10,681	235	NK	10,916	5,251,000	5,107,300	10,865	121,919

[a] As shown in JRB from nine references.

[b] Not known.

[c] Estimated by Arthur D. Little, Inc.

[d] Based on % yield and including amount destroyed in treatment of wastewater.

When the amounts of benzene released, carried over and destroyed, were summed and subtracted from the amount used as feedstock, the difference was 121,904 kkg. This amount, an artifact of inexact release rates, etc., is the amount unaccounted for and will include the small fraction of benzene disposed onto land in solid wastes resulting from these processes. The land releases are not expected to be significant.

3.5.2 Nonconsumptive Use

In 1978, <5% benzene production was used nonconsumptively; i.e., benzene was not converted to another compound before use. The categories of nonconsumptive use and the estimated amounts used are as follows:

Use	Benzene Used
Solvent	(kkg)
Pesticide	Unknown

3.5.2.1 Solvent Use

Solvent use of benzene has decreased since the 1977 OSHA Emergency Benzene Standard and the 1977 ban on the use of benzene in consumer goods by the Consumer Products Safety Commission (Neufeld et al. 1978).

Neufeld et al. (1978) reported on the use of benzene as a solvent (9600 kkg in 1978) and the releases associated with this use. They estimated that benzene solvent was either released or destroyed by industrial emission control processes. The fraction released was estimated from information on control systems obtained during interviews with representatives of companies using benzene as a solvent. JRB used data from Hillman et al. (1978) to estimate releases due to benzene in consumer products. It was assumed that all of this benzene was released to air except benzene in the "home fuels" category, which was destroyed (see Table 3-11). These authors also documented the effect of the 1977 OSHA and CPSC actions on benzene use: estimated losses of benzene due to solvent use were 600-700 kkg in 1976 and only 2500 kkg in 1978. Cyclohexane is replacing benzene in many solvent uses.

When used as a solvent in industrial processes, benzene may be released through evaporation or in effluent discharges. In general, each of these processes has a characteristic ratio of air to water releases. However, because of the range of ratios possible -- from 50:50 to 100% of air emissions -- it was not possible to quantify the total amount of benzene released to each medium as a result of solvent use. This rationale was also used in Section 3.4.3 for contaminated solvents.

Releases of benzene due to disposal of solid residues were not quantifiable; however, these releases are considered small. The rate

TABLE 3-11. ESTIMATED AIR RELEASES OF BENZENE FROM USE AS A SOLVENT, 1978

Solvent Use	Amount Used (kkg)	Amount Destroyed (kkg)	Releases (kkg)
General organic synthesis	7,400	6,400	1,000
Pharmaceutical synthesis	730	510	220
Small volume chemicals			
Aluminum alkyls	1,000	0	1,000
Alcohols	330	150	180
Consumer products	130[a]	20	110
Total	9,590	7,080	2,510

---

[a] Estimate applies to 1977.

Sources: Neufeld et al. (1978), Hillman et al. (1978).

of production of benzene-containing residues, the percentage of benzene (by weight) in the residues, and the method of residue disposal are required to evaluate land releases of benzene from solid wastes.

3.5.2.2 Pesticide Use

The U.S. EPA Pesticide Product Information File lists seven products (mostly screw worm pesticides) containing benzene. The percentage of benzene in each product is also given; however, the amount of each product formulated per year was not available. Thus, total benzene used for this purpose could not be quantified. Screw worm killers are not a major part of pesticide sales.

3.6 TRANSPORTATION AND STORAGE OF BENZENE

Releases occur when benzene is moved from producers to users. The releases described in this section are distinct from those described in the section related to gasoline, which also includes losses due to storage and transportation, loading, and storage. Ninety-nine percent of environmental releases of benzene are to air, with the remainder to water as a result of barge transportation of benzene. JRB did not mention leaks or spills onto land. A small amount of benzene is probably released to land from transfer or other operations.

Benzene releases due to storage are classified as standing and withdrawal losses. The factors mentioned previously, as well as the length of storage time cause storage standing losses. Withdrawing benzene from the tank increases the amount lost; usually, this is from the evaporation of benzene retained on the sides of the tank as the roof sinks (PEDCo 1977). Based on release factors of PEDCo (1977) and SRI (1978), the amount of benzene estimated as air losses during storage is between 105 and 4900 kkg.

Benzene is transported by railroad tank car, tank trucks, barges on inland waterways, and pipelines. Generally, before benzene is transported, it is first collected and temporarily stored in a "rundown tank", where it is inspected for product quality. Then, it may be transferred to two sets of shipping tanks, one for railcar and truck loading and the other for barge loading. The rail and truck loading tank is also used to feed pipelines. Benzene losses from these tanks may be characterized as standing losses (caused by evaporation around perimeter roof seals) and withdrawal losses (caused by emptying the tank). Based on the release factors of Dunavent (1978), the air release caused by loading to transport vehicles is 1300 kkg, assuming all stored benzene is passed through rundown tanks.

To estimate transportation-associated releases, JRB assumed that 50% of the transport takes place by rail or truck, and the rest occurs by barge. Based on the release rates of SRI (1978), total releases to air are 980 kkg.

Thus, the total loss to air that occurs as a result of conveying noncaptive benzene from producer to consumer is between 2400 and 7200 kkg. JRB estimated water losses as 10% of air releases and are thus 24-72 kkg.

3.7 SUMMARY

Total environmental releases of benzene are estimated to be about 246,080 kkg/yr, which is approximately 5.0% of total U.S. production.

Although the total amount of benzene disposed on land has not been estimated completely, it is clear that the amount accounted for (394 kkg) is insignificant, compared with air emissions (234,945 kkg), while water discharges are about one order of magnitude more (1,131 kkg).

The estimated releases of benzene to the environment, from both sources and users, are summarized in Table 3-12.

The largest contributors to environmental releases of benzene are in the indirect source category. Petroleum refining is the source of the major land-destined wastes, while fuel combustion generates the bulk (72%) of known air emissions. Petroleum refining, gasoline transport and storage, and use as a chemical feedstock contribute 21% of the quantified releases.

The remaining land sources, which have not been estimated because of insufficient data, are probably small and would not add significantly to the small amount documented.

Water discharges are comparatively small and the largest known source (55%) occurs as a result of production refining. Washwater from solvent use in both home and industry will likely be discharged to public sewer systems and thus be received for treatment by POTW's. However, it was not possible to calculate a figure for releases industrywide.

The benzene materials balance is shown in Figure 3-3. The total amount of benzene available for distribution in the environment was 11,121,274 kkg in 1978 (see Table 3-1). Of this amount, 26,992 kkg was released to the environment at the source, leaving 11,094,282 kkg available for use or distribution. Of this amount, 99.7% (11,059,380 kkg) was accounted for in use as feedstock, solvent, export, and in fuels consumed. The remainder, 34,902,is an unaccounted for discrepancy between production and use. Of the amount used, 96.6% is destroyed (or physically removed from the United States by exports (1.4% of total use), and 2.4% is released to the environment.

TABLE 3-12. SUMMARY OF ANNUAL ENVIRONMENTAL RELEASES OF BENZENE

Source	Maximum Estimated Releases (kkg)			Total
	Air	Water	Land	
Direct Sources				
Refining Production	3,139	620	141	3,900
Coke Plant Production[a]	768	10	8	786
Exports	?	2	15	17
Imports	13	13	0	26
Transport and Storage	7,200	72	?	7,272
Indirect Sources				
Coal Coking	1,872	?	?	1,872
Petroleum Refining	20,000	1	230	20,230
Gasoline Combustion	130,059	0	0	130,059
Gasoline Transport and Storage	21,000	0	?	21,000
Use of Other Fuels	40 213	?	?	40,213
Oil Well Drilling	?	?	?	?
Oil Spills	0	30	0	30
Use of Contaminated solvents	?	?	?	7,100
Resource Mining and Processing	0	148	0	148
Uses				
Chemical Feedstock	10,681	235	?	10,916
Solvent	?	?	0	2.510
Pesticide	?	?	?	?
TOTAL	234,945[b]	1,131[b]	394	246,080

[a] Released from coking operations only. Releases due to light oil refining not estimated.

[b] Subtotal does not include releases due to solvent use because the ratio between air and water was not quantified.

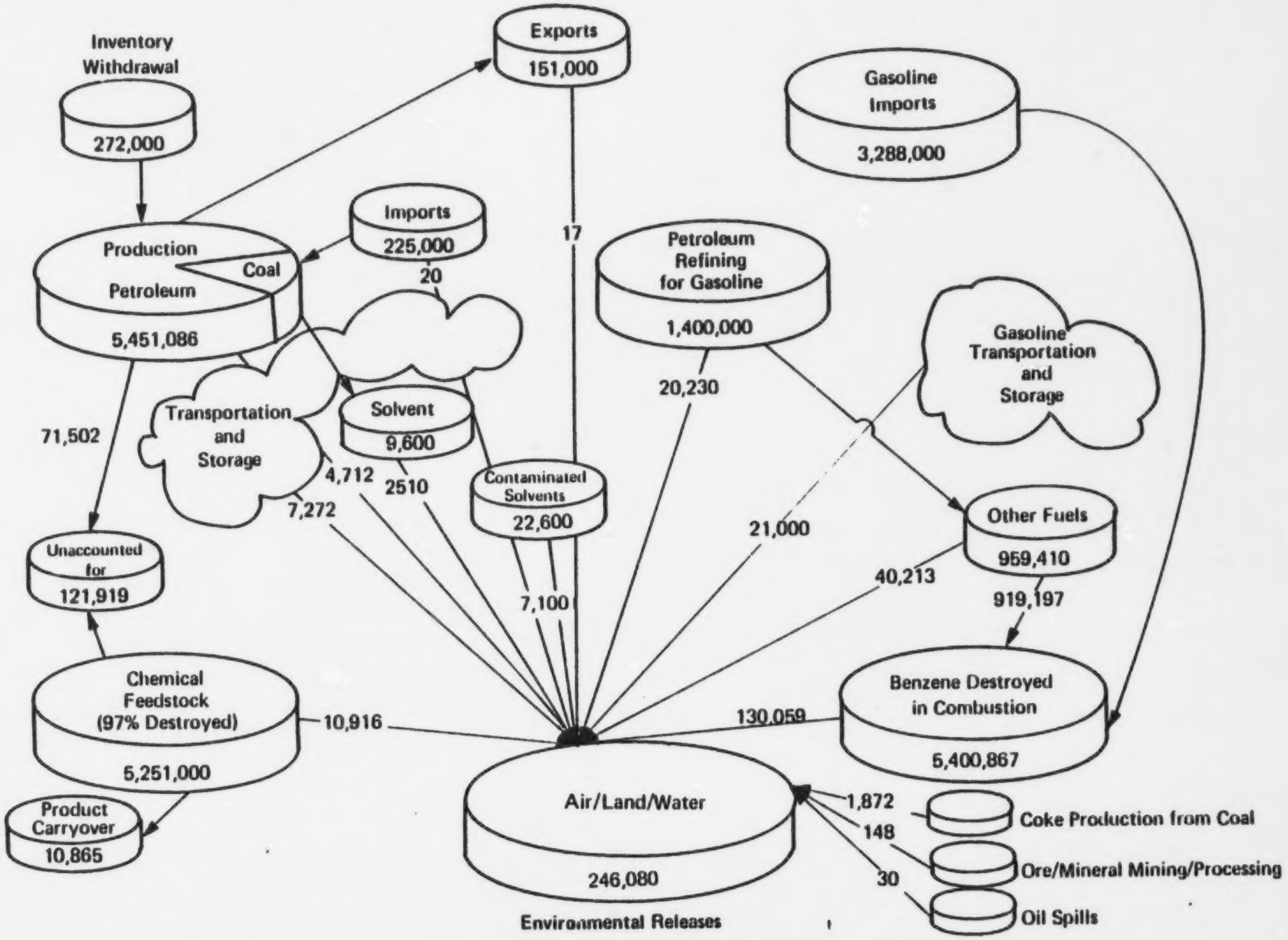
Inventory Withdrawal
272,000
Exports
151,000
Gasoline Imports
3,288,000
Imports
225,000
Production
Coal
Petroleum
5,451,086
20
17
Petroleum Refining for Gasoline
1,400,000
Gasoline Transportation and Storage
Transportation and Storage
Solvent
9,600
71,502
4,712
2510
7,272
Contaminated Solvents
22,600
20,230
21,000
Other Fuels
959,410
Unaccounted for
121,919
7,100
40,213
919,197
Chemical Feedstock (97% Destroyed)
5,251,000
10,916
130,059
Benzene Destroyed in Combustion
5,400,867
Product Carryover
10,865
Air/Land/Water
246,080
Environmental Releases
1,872
148
30
Coke Production from Coal
Ore/Mineral Mining/Processing
Oil Spills

FIGURE 3–3 MATERIALS BALANCE FOR BENZENE
(Areas are approximately to scale)

In the balance for benzene used as feedstock alone, 121,191 kkg were unaccounted for. Some portion of this amount is the volume disposed onto land. Thus, environmental releases are slightly underestimated. Consequently, in 1978, the total amount of unaccounted for benzene in the materials balance is 1.1% of the amount used (11,059,380).

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4.0 FATE AND DISTRIBUTION OF BENZENE IN THE ENVIRONMENT

4.1 INTRODUCTION

The following description of the environmental fate of benzene is based on critical pathway analysis. Resultant concentrations were estimated, where possible, and the distribution of benzene in the environment is described.

In this approach, the benzene releases from all known sources to the three physical media are traced through the environment to the human and biotic receptors. First, data on the physical, chemical, and biological characteristics of benzene are reviewed. The pathways and processes that result in transfer of benzene from one medium to another are analyzed. This includes a simple "partitioning" model, supplemented by consideration of the relative rates of the transfer processes. The next step is to evaluate major fate processes, such as chemical transformation, that may occur in the media of interest in which benzene is most likely to reside. The processes considered include chemical transformations and biodegradation within the media of interest.

The loading rates, intermedia transfer processes, and intramedia transformation/degradation processes are then used to estimate probable ranges of concentration of benzene in the environmental media. This is done by calculations involving "single compartment" models and by the U.S. EPA's EXAMS (Exposure Analysis Modeling System) model. The final step is to summarize the critical pathways, ranges of concentrations of the pollutant in environmental media, and compare these with the available monitoring data.

For benzene, the results of the materials balance analysis indicate that environmental releases are predominantly to the air, 95.5% (234,945 kkg) the remaining 0.3% is discharged to water, 0.2% is disposed of on land, and 3.8% were not assigned to any media.

The fate of benzene released to all three environmental media--air, soil, and water--is considered in the critical pathways analysis of this section.

4.2 PHYSICAL, CHEMICAL, AND BIOLOGICAL CHARACTERISTICS OF BENZENE

Benzene is an organic chemical whose physical and chemical characteristics have been thoroughly documented. Table 4-1 summarizes the physicochemical data that are directly relevant to the partitioning and movement of benzene in the environment, and additional basic information on properties of the bulk chemical that may be useful in evaluating particular situations (e.g., spills).

Benzene is moderately volatile as evidenced by the equilibrium vapor pressure of 0.13 atm at 26°C. However, the relatively high water

TABLE 4-1. PROPERTIES OF BENZENE RELATED TO ENVIRONMENTAL DISTRIBUTION

Property	Value	Reference
Molecular Formula	C_6H_6	
Molecular Weight	78.12	
Melting Point, °C	5.5	Weast (1979)
Boiling Point, °C	80.1	Weast (1979)
Water Solubility, mg/l	1800 at 25°C	Howard and Durkin (1974)
	1780 at 25°C	Mackay and Leinonen (1975)
	1750 at 10°C	Mackay and Leinonen (1975)
	820 at 22°C	Chiou *et al.* (1977)
Vapor Pressure, Torr	1 at -36.7°C	Weast (1979)
	10 at -11.5°C	
	40 at 7.6°C	
	100 at 26.1°C	
	400 at 60.6°C	
	760 at 80.1°C	
	45 at 10°C	Mackay and Leinonen (1975)
	95 at 25°C	
Saturated Vapor Concentration, g/m^3 (%v:v)	193 (5.9) at 10°C 407 (12.5) at 25°C	Calculated from vapor pressure
Octanol:Water Partition Coefficient	135 at 25°C	SRI (1980)
Sediment:Water Partition Coefficient (K_{oc})	74 at 25°C	SRI (1980)
Bioconcentration Factor (K_p)	22 at 25°C	SRI (1980)

solubility (1.8 g/l) indicates that aqueous media are also important. The low values of the partition coefficients and bioconcentration factor in Table 4-1 suggest that benzene is less likely to accumulate in soil, sediment, or biotic environmental compartments than in air or water.

The chemical reactivity of benzene is low, primarily because of the unique resonance stability of the benzene ring system (Hendrickson et al. 1970). Several authors have noted that extremely high temperatures, pressures, and/or reagent strengths are required to accomplish the commercially important oxidations or electrophilic substitution reactions of benzene (Howard and Durkin 1974, Walker 1976, Mara and Lee 1978). Benzene is essentially unreactive under mild-to-moderate chemical (thermal) reaction conditions.

However, the benzene ring can be attached by highly reactive species, such as hydroxyl radical or ozone, which may be present in the environment. Reaction with hydroxyl radical is an important environmental fate process.

Biological degradation of benzene can be accomplished by both individual species and mixed microbial populations isolated from various environmental media. No microorganism is known, however, that can directly cleave the unsubstituted benzene ring (Howard and Durkin 1974).

Biodegradation is indicated as a possibly important intramedia fate process for both soil and water systems. Proper conditions to support these processes must hold, however.

4.3 MONITORED LEVELS IN THE ENVIRONMENT

Monitoring data for concentrations of benzene in the environment have been collected for air, water, soil, and foodstuffs. Because of the concern over inhalation, particularly in occupational situations, the most extensive data base covers benzene in the air.

4.3.1 Air

Background levels of benzene are typically between 1.0-3.5 $\mu g/m^3$ in areas that are remote from sources. Air concentrations fall in the range 4 (Conneman 1978) to 160 $\mu g/m^3$ (Altshuller 1969) for ambient urban air where the number of sources (i.e., chemical plants, cars) increases, compared with 4.5 $\mu g/m^3$ measured at residential areas isolated from traffic (Battelle 1979). Table 4-2 contains air concentrations for various locations and types of sites. Battelle (1979) performed a major study of various sites in Columbus, OH, which indicated a direct correlation between traffic volume and benzene levels in the air. Traffic counts were available at only one site, however (a highway). These counts were made at hourly intervals and compared

TABLE 4-2. LEVELS OF BENZENE IN AIR

Site Description	Benzene (μg/m³) Mean	Benzene (μg/m³) Range	Reference
Urban Locations			
Denver, CO	9.6	95.8 max	Ferman *et al.* (1977)
Houston, TX	-	4-48	Bertsch *et al.* (1975)
Los Angeles Basin, CA	122	64-192	Altshuller and Bellar (1971)
Columbus, OH	118	?-412	Battelle (1979)
Midtown Intersection	12.3	5.9-21.3	
Highway:			
Eastbound (15,769 cars/24 hr)	9.6	5.6-14.2	
Westbound (20,963 cars/24 hr)	23	13.8-35.9	
Residential--24-hour average	5.1	3.2- 8.1	
Nighttime (background)	4.5		
Gasoline Station Vicinities[a]			
Within 300 m of 4 Stations	3.1	0.5-13.7	
Within 300 m of 2 Stations	2.3	0.9- 4.5	
Within 200 m of 1 Station	2.4	0.6- 5.0	
Within 200 m (general)		9.6-32	API (1977)
Other Locations			
Within 200 m of Rural Gasoline Stations		1.3-11.8	API (1977)
NJ		trace-300	RTI (1977)
CA	21.7	18 -34	RTI (1977)
Background - Remote Areas in U.S.	2.2	1.0- 3.5	Washington State Univ. (1973)
Edison, NJ, Landfill Site	900,000		RTI (1976)
Downwind from Landfill		10 -1550	RTI (1976)
Upwind from Landfill		trace- 200	RTI (1976)

[a] Data from up to seven monitors were included in the average for each site.

with benzene concentration measurements taken concurrently; the highest levels were recorded during morning and evening rush-hour traffic, and the lowest levels were recorded late at night, during periods of low-traffic density. The eastbound traffic had a lower overall traffic density (15,769 vehicles/24 hr) and an average benzene level of 9.6 $\mu g/m^3$. The westbound traffic, with a higher vehicle count (20,963/24 hr) had a higher average benzene level of 23 $\mu g/m^3$. Further research is required to estimate emission rates from traffic of various vehicle mixtures, road conditions, street grid patterns, and other sources.

Battelle (1979) did not sample the air directly at gasoline service stations; however, monitors were placed within 300 m of such sources. Higher levels were recorded at the intersection with 4 stations (3.1 $\mu g/m^3$) as compared with intersections with only one or two stations (2.3-2.4 $\mu g/m^3$). This difference is small and cannot be interpreted without accompanying traffic data. Levels near rural gasoline stations fall within the same range (1.3-11.8 $\mu g/m^3$) as does the air in urban service station vicinities (<1-32 $\mu g/m^3$).

The air near several chemical plants and petroleum refineries that use benzene has been sampled; the results of these studies are shown in Table 4-3. The range of benzene levels in the air was 1.0-111.2 $\mu g/m^3$ for chemical plants and 3.2-824.1 $\mu g/m^3$ for refineries. More typically (the mean value after eliminating outliers), levels at these facilities are about 14 $\mu g/m^3$ for chemical plants and 9 $\mu g/m^3$ for refineries. Although these means indicate slightly higher levels at chemical plants, the latter source type appears to have the potential for higher local benzene concentrations than the former because of the larger volume of benzene being handled.

The benzene concentrations measured in the air at occupational stations (i.e., foreman, still operators, pump operators, etc.) at nine chemical plants and five service stations are presented in Table 4-4. The table shows the mean for each type of plant or service station study and the reported range of concentrations. At chemical plants, the typical concentrations that would be breathed by workers are in the range of 2000-10,000 $\mu g/m^3$. Maximum levels are about 30 for many processes; however, they may reach nearly 500,000 $\mu g/m^3$.

At service stations, however, the levels of benzene in the air are much lower by comparison. Battelle (1979) and Runion (1977) recorded averages <1000 $\mu g/m^3$ and a maximum of 5400 $\mu g/m^3$.

4.3.2 Water

Information on benzene levels for water have been recorded by several studies and retrieved from the U.S. EPA's STORET Water Quality System (September 1981).

TABLE 4-3. LEVELS OF BENZENE IN AIR NEAR CHEMICAL PLANTS AND PETROLEUM REFINERIES

Description Chemical Plants	Benzene (μg/m^3) Mean	 Range	Source
Nitrobenzene Plant, WV	8.78	1.3-22.4	Battelle (1979)
NJ, Nitrobenzene	8.9		RTI (1977)
LA, Nitrobenzene	1.9		RTI (1977)
Cumene Plant, PA	40.0	3.8-111.2	Battelle (1979)
Maleic Anhydride Plant, TN	23.16	8.3-52.4	Battelle (1979)
Maleic Anhydride Plant, WV[a]	12.5	1.0-94.9	Battelle (1979)
NJ, Maleic Anhydride	2.9		RTI (1977)
Detergent Alkylate Plant, CA	6.4	3.2-9.6	Battelle (1979)
WV, Detergent Alkylate	108.6		RTI (1977)
Benzene Plant, LA	20.1	1.9-43.1	Battelle (1979)
TX, Ethyl Benzene Styrene	44.7		RTI (1977)
Coke Ovens, PA	9.3	1.3-39.3	Battelle (1979)
LA, Phenol	2.9		RTI (1977)
TX, (Unknown)	2.6		RTI (1977)
Petroleum Refineries[b]			
Mid-Atlantic	9.6		API (1977)
Pacific N.W.	6.4		API (1977)
Midwest	<3.2		API (1977)
Gulf Coast	16.0		API (1977)
Missouri	230.0		RTI (1977)
Texas	10.9		RTI (1977)
California	824.1		RTI (1977)

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[a] Near a coking facility and a refinery.

[b] All samples within 1 kilometer of plants.

TABLE 4-4. LEVELS OF BENZENE IN AIR FOR HUMAN ACTIVITIES

Type of Industrial Plant	Benzene (mg/m^3) Mean	Benzene (mg/m^3) Range	Reference
Coke Plant with Benzene Refining	-	1.6-96	NIOSH (1974)
Ethylbenzene Plant	2.9	2.7-10.7	NIOSH (1974)
Benzene Recovery Plant	4.2	2.7-10.7	NIOSH (1974)
Cumene Plant	-	0-21.5	NIOSH (1974)
-- Caustic Addition	-	5.4-32.2	NIOSH (1974)
Aniline Production	-	0.32-1.1	NIOSH (1974)
-- Benzene Unit	-	0-483.3	NIOSH (1974)
Chlorobenzene Production	-	0.54-33.3	NIOSH (1974)
Alkyl Benzene Production	-	1.6 -78.9	NIOSH (1974)
Benzene Light Oil Plant	-	2.7-268.5	NIOSH (1974)
Benzol Plant Operator	-	18-64[a]	NIOSH (1974)
Service Stations			
Customer Areas	0.86	0.38-5.4	Battelle (1979)
Attendant Areas	0.26	0.11-0.39	Hartle and Young (1976)
Attendant Areas	0.26	0.29-0.58	
Attendants - charcoal tube samples	1.02	0-5.4	Runion (1977)
General Air	0.44	0.17-0.66	NIOSH (1974)

[a] Ten minute time-weighted averages.

4.3.2.1 Drinking Water

Few data are available on benzene levels in drinking water. It was identified by the U.S. EPA (1977) in 21.6% of finished drinking water supplies taken from surface water and in 60% of all supplies tested (Coniglio et al. 1980). Median benzene concentrations for all NOMS data were <2 µg/l. A study of priority pollutants in tap water from St. Louis, Atlanta, Cincinnati, and Hartford revealed no benzene at detection limits of 1 µg/l and an estimated recovery factor of 89% (Levins et al. 1979). The U.S. EPA (1972) found trace amounts of benzene in finished tap water taken from the Mississippi River.

4.3.2.2 Ambient Water

The U.S. EPA's STORET system includes data from 185 ambient water quality monitoring stations. As of September 1981, the total number of samples on record was 889, of which 156 were unremarked. Unremarked data are those for which an accurate (within the testing equipment limitations) reading of the concentration is given. These are generally accepted at face value, while remarked data are regarded as upper limits.

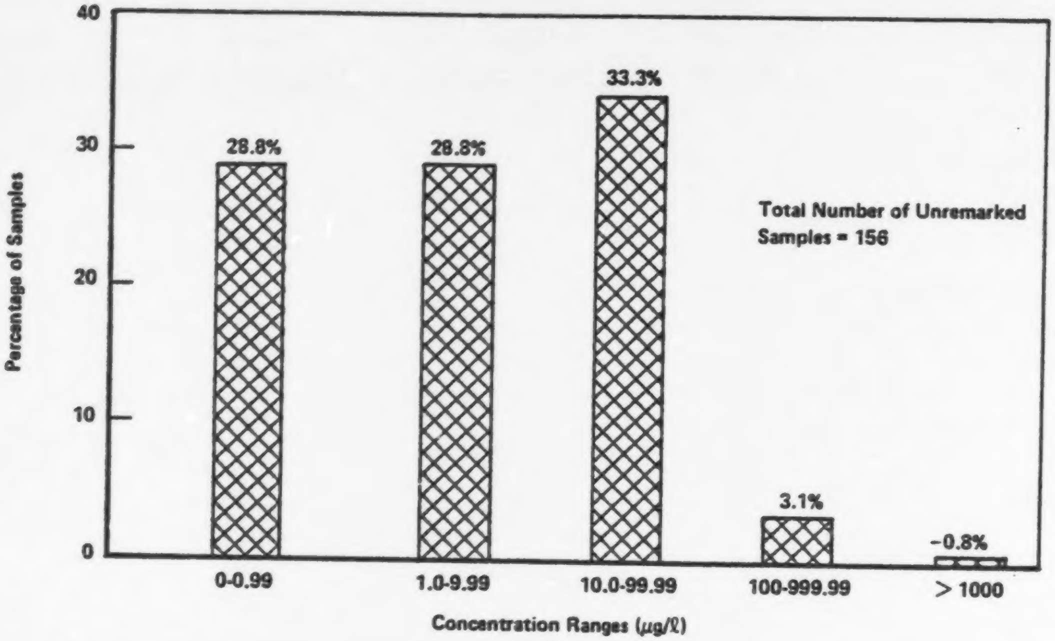
Less than 20% of the samples for ambient benzene fall into the unremarked category. The distribution shown in Figure 4-1, is flat up to 100 µg/l, with approximately one-third of the data in each of the concentration ranges: 0-1, 1-10, and 10-100 µg/l. Only 4% of the samples were above 100 µg/l and <1% were above 1000 µg/l. The distribution of the remarked samples is shown in Figure 4-2. Ninety percent of these values were below 100 µg/l, a result similar to the unremarked data. The skew in the histogram towards the 1-100 µg/l range is probably due to a predominance of benzene tests with detection limits of 10 µg/l and 100 µg/l.

Table 4-5 records the ambient benzene levels by major water basin. The median (50% level) values were 10 µg/l or lower for 15 of the 18 basins, and was 5 µg/l for the country as a whole. The highest maximum level recorded was 1260 µg/l, an unremarked value from the California Basin. Levels above 100 µg/l were shown in the Ohio River Basin (140 µg/l), the Lake Michigan Basin (310 µg/l) and the Lower Mississippi Basin (210 µg/l).

This data set offers evidence that levels of benzene in ambient water is <10 µg/l ∿50% of the time and <100 µg/l ∿95% of the time. Rarely have levels above 100 µg/l been documented. The Lake Michigan and Lower Mississippi Basins are the two showing the highest levels, probably due to the quantity and type of industrial activity prevalent in these areas.

Levels of benzene documented for POTW systems in six cities in Table 4-7 ranged from <1 to 143 µg/l in influent water. The average percent removal of benzene during treatment was 90% with 3 of 5 plants achieving ∿100% removal. These data also show that benzene is concen-

FIGURE 4-1 DISTRIBUTION OF UNREMARKED BENZENE CONCENTRATIONS BY AMBIENT WATER QUALITY STATIONS, 1977-81



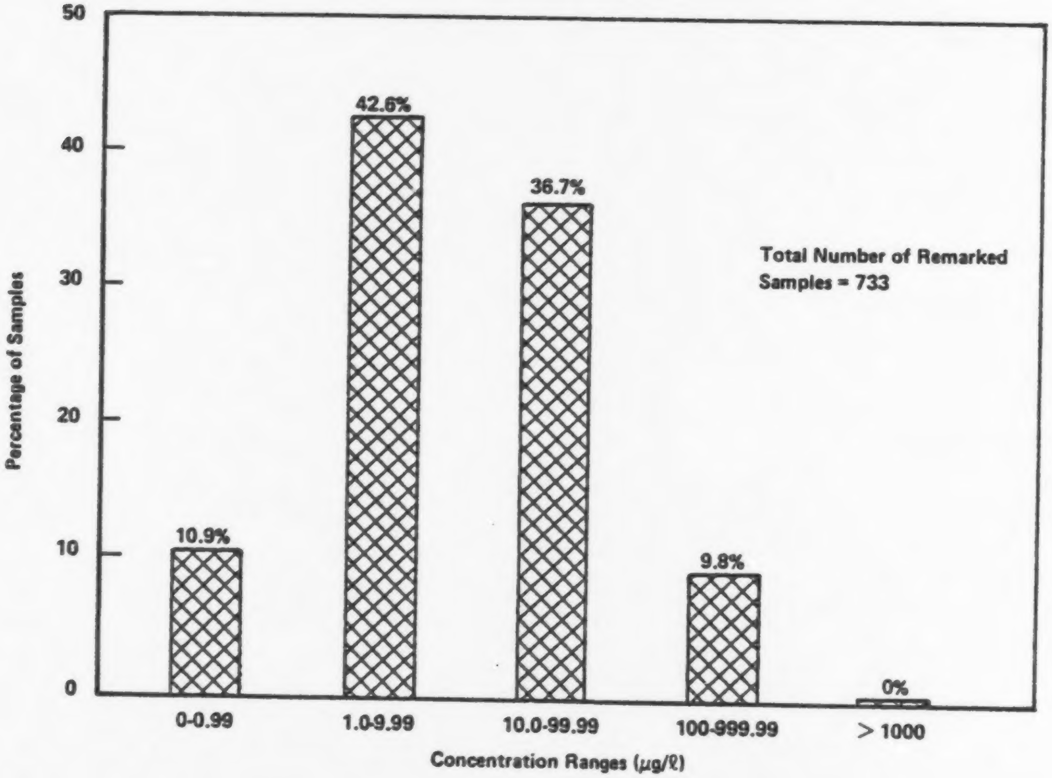


FIGURE 4-2 DISTRIBUTION OF REMARKED BENZENE CONCENTRATIONS BY AMBIENT WATER QUALITY MONITORING STATIONS, 1977-81

Source: U.S. EPA (1981).

TABLE 4-5. CONCENTRATIONS OF BENZENE BY MAJOR BASIN IN 1980

Basin Name	No. of Samples	Mean Concentration (µg/1)	Median Concentration (µg/1)	Maximum Concentration (µg/1)
1. Northeast				
REM[a]	1	10.0	10.0	10.0
UNREM[b]	0	-	-	-
2. North Atlantic				
REM	36	0.0	0.0	0.0
UNREM	6	0.9	0.7	2.5
3. South East				
REM	13	6.5	7.5	10.0
UNREM	10	0.0	0.0	0.0
4. Tennessee River				
REM	49	6.5	5.0	10.0
UNREM	4	12.5	13.0	17.0
5. Ohio River				
REM	27	9.4	10.0	10.0
UNREM	7	51.0	15.0	140.0
6. Lake Erie				
REM	9	10.0	10.0	10.0
UNREM	0	-	-	-
7. Upper Mississippi				
REM	11	46.4	50.0	50.0
UNREM	0	-	-	-
8. Lake Michigan				
REM	9	5.9	5.0	10.0
UNREM	5	187.2	220.0	310.0
9. Missouri River				
REM	32	50.0	50.0	50.0
UNREM	0	-	-	-
10. Lower Mississippi				
REM	16	4.0	5.0	5.0
UNREM	16	67.7	60.0	210.0

---

[a] REM = Remarked Samples
[b] UNREM = Unremarked Samples

TABLE 4-5. CONCENTRATIONS OF BENZENE BY MAJOR BASIN IN 1980 (Continued)

Basin Name	No. of Samples	Mean Concentration (μg/l)	Median Concentration (μg/l)	Maximum Concentration (μg/l)
11. Colorado River				
REM	43	8.8	10.0	10.0
UNREM	0	-	-	-
12. Western Gulf				
REM	18	10.0	10.0	10.0
UNREM	0	-	-	-
13. Pacific Northwest				
REM	13	0.0	0.0	0.0
UNREM	0	-	-	-
14. California				
REM	34	5.1	5.0	10.0
UNREM	49	57.8	6.0	1260.0
15. Great Basin				
REM	4	10.0	10.0	10.0
UNREM	0	-	-	-
16. Lake Huron				
REM	11	10.0	10.0	10.0
UNREM	0	-	-	-
17. Lake Superior				
REM	5	10.0	10.0	10.0
UNREM	0	-	-	-
18. Hudson Bay				
REM	4	0.0	0.0	0.0
UNREM	0	-	-	-
19. United States[c]				
REM	677	18.9	5.0	100.0
UNREM	129	46.1	6.8	1260.0

---

[c] All samples, 1978-1981

TABLE 4-6. LEVELS OF BENZENE IN WATER NEAR AND IN EFFLUENTS FROM CHEMICAL PLANTS

Sample Site Description	Benzene (μg/l) Mean	Range	Reference
Water Near Discharges			
Ohio R. - Nitrobenzene Plant - downstream	4.1	2.6-6.3	Battelle (1979)
Nitrobenzene Plant - upstream	12	9.2-15.4	Battelle (1979)
Cumene Plant - PA	1.4	0 - 2.4	Battelle (1979)
Maleic Anhydride Plant, TX - upstream	<1	-	Battelle (1979)
Anhydride Plant, TX - downstream	2	-	Battelle (1979)
Detergent Alkylate Plant, CA	<1	-	Battelle (1979)
Benzene Plant, LA - upstream Miss. R.	2	-	
Benzene Plant, LA - downstream Miss. R.	1	-	Battelle (1979)
Drinking Water - U.S. Cities		0.1-0.3	NCI (1977)
Drinking Water - U.S. Cities	0.2	0.1-0.3	U.S. EPA (1977)
Effluents			
Nitrobenzene Plant, WV	104	81-134	Battelle (1979)
Cumene Plant, PA	4.3	3.3-5.4	Battelle (1979)
Maleic Anhydride Plant, TX	8	-	Battelle (1979)
Benzene Plant, LA	179	-	Battelle (1979)
Seven Cities	2.2	0-5	Burns and Roe (1979)
Seven Cities - Sludge	61	12-171	Burns and Roe (1979)
POTW - effluent	1	-	Versar (1978)
POTW - sludge	400	-	Versar (1978)
Water upstream of POTW	ND	-	Versar (1978)
Water downstream of POTW	<1	-	Versar (1978)

Note:

ND = not detected.

TABLE 4-7. LEVELS OF BENZENE IN POTW SAMPLING DATA

<table>
<tr><th></th><th colspan="5">Benzene (μg/1)</th></tr>
<tr><th>Cities</th><th>Influent</th><th>Primary Sludge</th><th>Secondary Sludge</th><th>Final Effluent[b]</th><th>% Removal</th></tr>
<tr><td>Indianapolis, IN</td><td>143</td><td>171</td><td>10</td><td>(1)3</td><td>98</td></tr>
<tr><td>Cincinnati, OH</td><td>10</td><td>1</td><td></td><td>(1)3</td><td>70</td></tr>
<tr><td></td><td></td><td colspan="2">33[c]</td><td></td><td></td></tr>
<tr><td>Atlanta, GA</td><td><1</td><td></td><td>(20)[a]</td><td><1</td><td>--</td></tr>
<tr><td></td><td></td><td colspan="2">40[c]</td><td></td><td></td></tr>
<tr><td>St. Louis, MO</td><td>5</td><td>42</td><td>(5)</td><td>1</td><td>80</td></tr>
<tr><td>Pottstown, PA</td><td>1</td><td>12</td><td>(11)</td><td>0</td><td>100</td></tr>
<tr><td>Grand Rapids, MI</td><td>1</td><td></td><td></td><td><5</td><td>100</td></tr>
<tr><td></td><td></td><td colspan="2">95[c]</td><td></td><td></td></tr>
</table>

Source: Burns and Roe (1979).

[a] Digested sludge.

[b] Concentrations in prechlorinated effluent, otherwise, chlorinated effluent.

[c] Combined sludge.

trated in sludge. The levels in secondary or biologically active sludge are somewhat lower than in the primary (physically settled) and combined sludges, which may indicate removal of benzene by volatilization during aeration or by acclimated bacteria (biodegradation). The Versar, Inc. (1978) data in Table 4-6 also show this pattern.

In Table 4-8, the results of an Arthur D. Little, Inc., (Levins et al. 1979) study of wastewaters from various socioeconomic sectors of four cities are shown. Sewage from commercial neighborhoods averaged to 2.7 μg/l, industrial sewage was about 1.3 μg/l, while residential sewage contained close to no benzene at all. In all four cities tested, no benzene was detected in the tap water.

4.3.3 Soil

Very few data were available on benzene levels in soils. Levels ranging from 13 to 115 μg/kg were reported in soil samples taken in the vicinity of chemical plants (see Table 4-9) that produce or use benzene (Battelle 1979). No background data have been found.

4.3.4 Food

Benzene has been detected in fruits, nuts, vegetables, dairy products, meat, poultry, eggs, fish, and several beverages (see Table 4-10). It is theorized that it occurs naturally, possibly as a flavor component in all of these foods. Only a small number of these foods has been analyzed quantitatively. Eggs have the highest concentrations (500-1900 μg/kg), followed by haddock (100-200 μg/kg) and Jamaican rum (120 μg/kg). Butter, beef, lamb, mutton, veal, and chicken have <10 μg/kg benzene levels (when the meats are cooked). It is postulated that the increase in benzene levels observed following cooking meats is due to the breakdown of aromatic amino acids.

Total dietary intake is estimated conservatively at about 250 μg benzene/day (NCI 1977) (as used, conservative implies a high value).

4.3.5 Summary

Concentrations of benzene in the air, water, and soil are higher in close proximity to sources. Occupational levels of benzene in air are the highest observed for that medium, while chemical plant and refinery discharges contain the highest recorded aqueous benzene levels. As the distance increases from the benzene source, concentrations decrease to levels <1 μg/l in water and 1.0 μg/m^3 in air; "clean" soil levels have not yet been documented.

4.4 ENVIRONMENTAL FATE MODELING

4.4.1 Equilibrium Partitioning

As an initial step in hazard or risk assessments for toxic chemicals, in the planning of laboratory and field tests, and in the inter-

TABLE 4-8. LEVELS OF BENZENE IN INFLUENTS TO SEWAGE TREATMENT PLANTS

	Benzene (μg/l)				
	Tap	Type of Neighborhood			
Cities	Water	Residential	Commercial	Industrial	Influent
Cincinnati, OH	0	.15	5.4	-[a]	3.7
St. Louis, MO	0	.8	2.8	1.3	7.0
Atlanta, GA	0	0	0.43	1.2	0
Hartford, CT	0	0	2.1	-[a]	0

Source: Levins *et al*. (1979).

[a] Not sampled for this city.

TABLE 4-9. LEVELS OF BENZENE IN SOIL NEAR CHEMICAL PLANTS

Description	Benzene (µg/kg) Mean	Range	Reference
Nitrobenzene Plant, WV	-	2-51	Battelle (1979)
Cumene Plant, PA	37.3	18-73	Battelle (1979)
Maleic Anhydride Plant, TX	22	-	Battelle (1979)
Detergent Alkylate Plant, CA	115	51-191	Battelle (1979)
Benzene Plant	13	12-14	Battelle (1979)

TABLE 4-10. FOODS REPORTED TO CONTAIN BENZENE[a]

Fruits

Apple
Citrus Fruits
Cranberry and Bilberry
Currants
Guava
Pineapple
Strawberry
Tomato

Nuts

Filbert (roasted)
Peanut (roasted)
Macademia Nut

Vegetables

Beans
Leek
Mushroom
Onion (roasted)
Parsley
Potato
Soya Bean
Trassi (cooked)

Dairy Products

Butter (0.5)[b]
Bleu Cheese
Cheddar Cheese
Other Cheese

Meat, Fish and Poultry

Beef (cooked)[d] (2 to 19)[c]
Chicken (<10)[d]
Egg (hard boiled) (500 to 1900)[e]
Haddock (100 to 200)[f]
Lamb (heated) (<10)[d]
Mutton (heated) (<10)[d]
Veal (heated) (<10)[d]

Beverages

Cocoa
Coffee
Jamaican Rum (120)[g]
Tea
Whiskey

---

[a] Numbers in parentheses are concentrations in µg/kg. Data adapted from Van Stratten (1977) unless noted.

[b] Siek and Lindsay (1970).

[c] National Cancer Institute (1977).

[d] Merrit (1972).

[e] MacLeod (1977); MacLeod and Cave (1976).

[f] Irradiated and nonirradiated haddock, respectively.

[g] Liebich _et al._ 1970.

pretation of monitoring data, rough estimates of the pollutant's environmental distribution can often be made by simple inspection of the chemical's properties. Mackay (1979) proposed a simple approach based on the fact that the fugacity of the pollutant must be the same in all phases when the system is in equilibrium.

The approach proposed by Mackay (1979) is a three-tiered approach. In Level I (the approach used here), all environmental compartments (phases) are assumed to be directly or indirectly connected and at equilibrium. The compartments considered are air, surface water, suspended sediments, bottom sediments, soil and aquatic biota. The Level I calculations require that these compartments be roughly described (volumes, temperature, sediment and biota "concentrations," etc.). It is clear that the model output depends on the nature of the "environment" selected. The compartment-specific parameters chosen here (somewhat arbitrarily) are listed in Table 4-11. A schematic diagram of the selected environment is shown in Figure 4-3. The Level I calculations do not consider degradation, or transport into or out of the selected environment. A relatively small number of chemical-specific parameters (see Table 4-11) are required for equilibrium partitioning. To obtain an absolute estimate of the equilibrium concentrations in each phase, it is necessary to estimate the total amount of the chemical that is likely to be in the selected environment.[1] The amount here is 30 mole/km^2, or 30 moles in the environmental compartment here with a surface area of 1 km^2. This amount is equivalent to the U.S. environmental losses over a 15-day period, divided by the area of the 48 contiguous states. Implicit in the selection of this quantity is an assumed atmospheric half-life (resulting from oxidative destruction) of about 2 days in urban environments or 20 days in rural areas. Thus, a value of 15 day's loading was selected as a reasonable estimate of the environmental burden.

Mackay (1979) provides details of the calculation methods; thus, they are not repeated here. The results are presented in Table 4-12.

When the percentage distribution of the benzene across compartments is considered, it is obvious that the high volatility of benzene dominates the environmental partitioning for the Mackay model. More than 99.9% of the chemical is predicted to be in the air medium at equilibrium. Water and soil account for 0.02 and 0.03%, respectively, of the benzene loading at equilibrium. The Mackay approach predicts that only very small fractions of the total mass of benzene will be distributed in the sediments or the biota.

A slightly different perspective is obtained by considering the concentrations, rather than the total mass loadings, in the environmental media. For the arbitrary but not unreasonable, compartment-

---

[1] Note that predicted ratios of concentrations between two phases will not be affected by the number selected.

TABLE 4-11. VALUES OF THE PARAMETERS USED FOR LEVEL I CALCULATION OF EQUILIBRIUM CONCENTRATIONS OF BENZENE USING MACKAY'S FUGACITY METHOD

Chemical-specific Parameters (25°C)

Solubility (mg/l):	1800	
Vapor pressure (torr)	95	
Adsorption coefficient (K_p) for:		
Suspended sediments	3.0	Assuming 4% organic carbon in sediments and 1% organic carbon in soils
Sediments	3.0	
Soils	0.7	
Octanol/water partition coefficient	135	

(Used for estimating a bioconcentration factor for aquatic biota.)

Total amount of chemical in compartment: 30 moles/km^2

(Equivalent to total U.S. environmental losses over a 15-day period, divided by the area of 48 contiguous states.)

Compartment-specific Parameters[a]

Temperature: 25°

Concentrations (S) of suspended sediments: 10g/m^3

Concentrations (S) of soils and sediments: 2×10^6 g/m^3

Volume fraction (B) of aquatic biota: 50×10^{-6} m^3/m^3

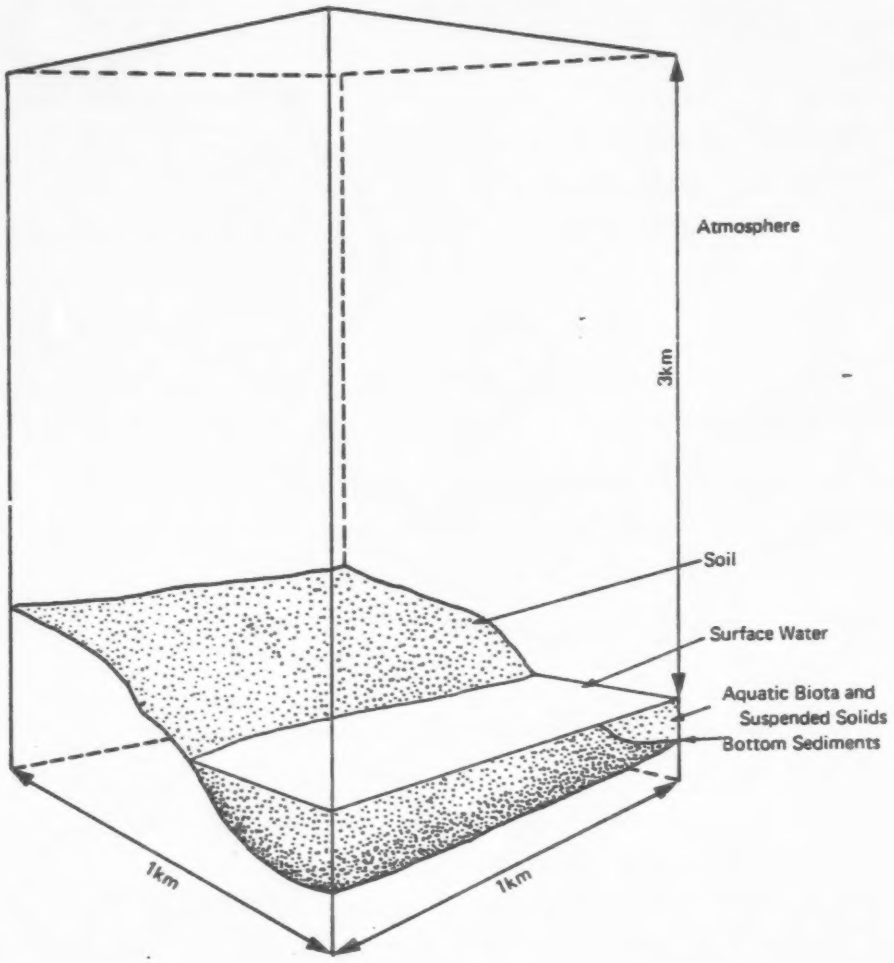
Fraction (y) of aquatic biota equivalent to octanol: 0.2

Accessible volume for each subcompartment:

Air	1 km x 1 km x 3 km (high) = 3×10^9 m^3
Surface Water	1 km x 0.05 km x 3m (deep) = 1.5×10^5 m^3
Sediments	1 km x 0.05 km x 10 cm (deep) = 5×10^3 m^3
Soils	1 km x 0.95 km x 14 cm (deep) = 1.3×10^5 m^3

Note:
In these preliminary calculations, the suspended sediments and aquatic biota are assumed to have the same "accessible volume" as the surface water compartment.

[a] Compartment-specific parameters may be selected to reflect the nature and size of any area of concern. The parameter values used here are not to be considered typical but reasonable.


Atmosphere
3km
Soil
Surface Water
Aquatic Biota and
Suspended Solids
Bottom Sediments
1km
1km

Note: Diagram is not to scale. Dimensions and accessible volumes of each subcompartment given in Table 4–11.

Source: Based on Mackay (1979).

FIGURE 4-3 SCHEMATIC OF ENVIRONMENTAL COMPARTMENT SELECTED FOR ESTIMATION OF EQUILIBRIUM PARTITIONING OF BENZENE

TABLE 4-12. EQUILIBRIUM PARTITIONING OF BENZENE CALCULATED USING MACKAY'S FUGACITY METHOD

Compartments	Z_i	V_i	f_i	M_i (moles)	C_i	% of Total Loading
Air	40	3×10^9 m^3	2.5×10^{-10}	30	0.78 ug/m^3	99.94
Water	185	1.5×10^5 m^3	2.5×10^{-10}	7×10^{-3}	0.0036 ug/l	0.02
Suspended Sediment	5.5×10^{-3}	1.5×10^5 m^3	2.5×10^{-10}	2×10^{-7}	0.010 ug/kg	-
Sediment	1.1×10^3	5×10^3	2.5×10^{-10}	1.3×10^{-3}	0.010 ug/kg	0.004
Aquatic Biota	2.5×10^{-1}	1.5×10^5	2.5×10^{-10}	8×10^{-6}	0.083 ug/kg	-
Soil	2.7×10^2	1.3×10^5	2.5×10^{-10}	8×10^{-3}	0.0024 ug/kg	0.03

Assumptions

K_{oc} = 74 (ug/kg)/(ug/l)   mw = 78

H = 229 (ug/m^3)/(ug/l) K_{ow} = 135 (ug/l)/(ug/l)   total benzene loading = 30 mol/km^2

Suspended solids   at 4% organic carbon content; concentration, 10 g/m^3

Sediments   at 4% organic carbon content; concentration, 2×10^6 g/m^3

Soil   at 1% organic carbon content; concentration, 2×10^6 g/m^3

Biota   20% equivalent to octanol; volume fraction 50×10^{-6} m^3/m^3

Definitions

Z_i = fugacity capacity constant for benzene in compartment i.

V_i = effective accessible volume of compartment i.

f_i = fugacity of benzene in compartment i. At equilibrium, fugacity in all compartments must be equal.

M_i = moles of benzene in compartment i.

C_i = concentration of benzene in compartment i.

specific parameters selected for this calculation, the water medium has an estimated concentration of 0.0036 μg/l. The equilibrium concentration in the air is calculated to be 0.7 $\mu g/m^3$ of benzene. Concentrations in the suspended and bottom sediments are calculated to be 0.01 μg/kg, about three times higher than those in the water. The calculated soil concentration is 0.0024 μg/kg; this is lower than for sediments because of the lower assumed carbon content of soil in this model. The concentration in aquatic biota was calculated to be 0.083 μg/kg of biomass, which illustrates benzene's moderate tendency to bioaccumulate.

4.4.2 EXAMS Modelling

The U.S. EPA's Exposure Analysis Modelling System (EXAMS) program is one approach to the integration of various intermedia transfer and intramedium transformation processes. The EXAMS model considers physical constants and reaction rate data for the chemical and the properties of typical and/or highly specific environments.

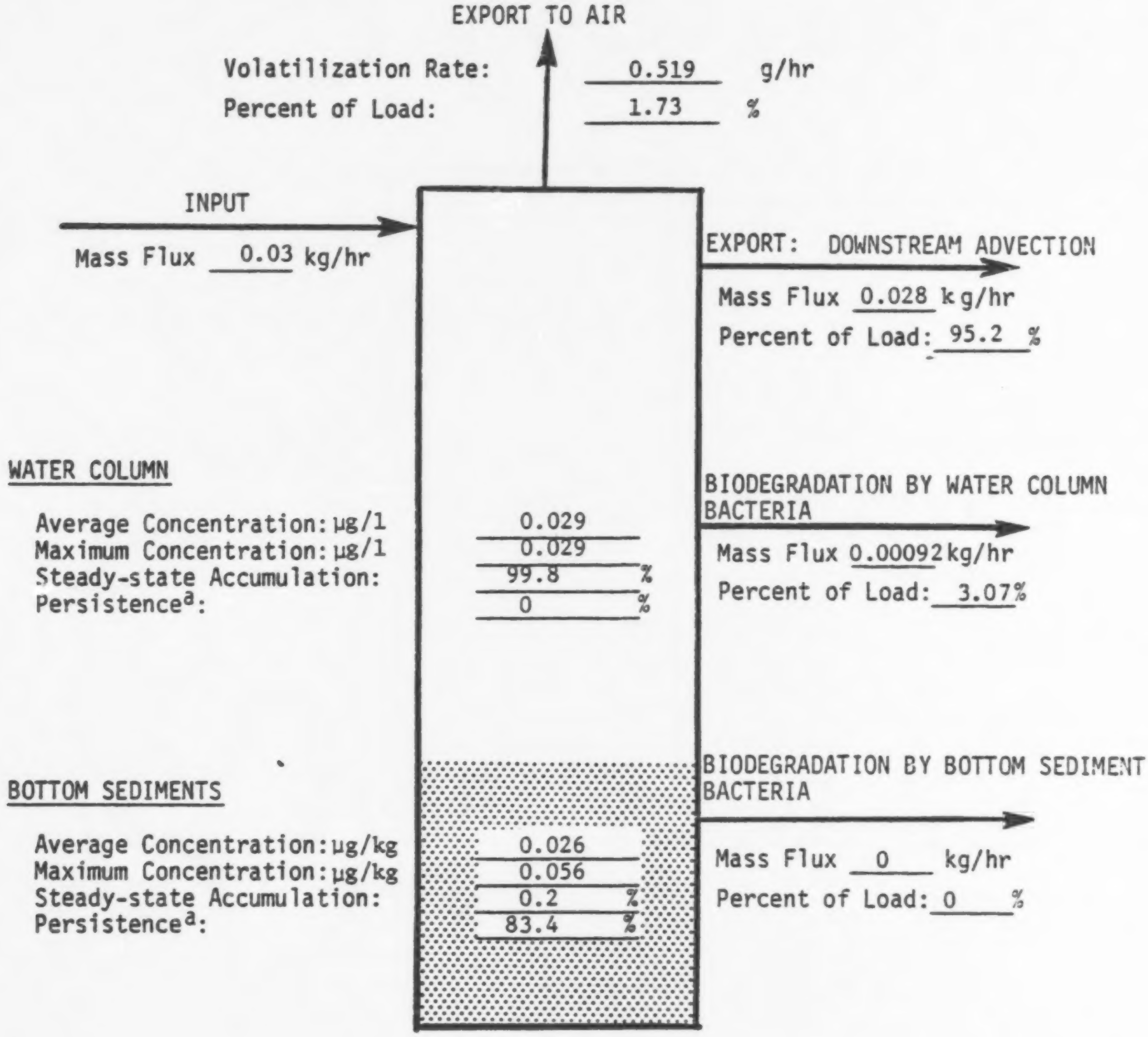
The environmental fate of benzene was modeled using four EXAMS scenarios: a "clean" river, a turbid river, an oligotrophic lake, and a eutrophic lake. Three loading rates were selected as inputs to EXAMS. The highest was 3.5 kg/hr based on a maximum effluent level measured for benzene at a petroleum refinery. The lowest rate was 0.002 kg/hr, which is also an effluent concentration for a refinery. The third rate, 0.03 kg/hr, was representative of benzene concentrations in both the textile industry and a small-scale coal-derived benzene production plant. This latter rate only was applied to all four scenarios. The extreme rates were only used for the riverine scenarios as the likely receiving water bodies. The results of the extreme inputs are given in Appendix B. (The results will scale directly with the loading rate until/unless the water solubility is exceeded or some other environmental compartment saturates.) The fundamental difference between the river and lake scenarios is that the former are flowing systems so that downstream transport/dispersal appears as a major fate process. The turbid river has a fivefold higher level of suspended sediment than the "clean" river.

The eutrophic lake differs from its "clean" counterpart in that it has much higher (three orders of magnitude) bacterial populations, as well as somewhat higher levels of sediment.

Schematic summaries of the results using EXAMS for these four scenarios are presented in Figures 4-4 through 4-7. In the river systems, downstream export appears as the dominant fate process and as 95.2% of the load. Volatilization is also a significant transport process, accounting for loss of 1.7% of the load within the ∿ 20-minute residence time of the river "slice" (see Figures 4-4 through 4-7). In the oligotrophic lake system, the relative importance of export and volatilization are reversed; volatilization accounts for 95% of the load and export for approximately 4%. In the eutrophic

FIGURE 4-4. RESULTS OF EXAMS MODELING OF THE ENVIRONMENTAL FATE OF BENZENE IN A TURBID RIVER

Ecosystem: Turbid River: Benzene



EXPORT TO AIR

Volatilization Rate: 0.519 g/hr

Percent of Load: 1.73 %

INPUT

Mass Flux 0.03 kg/hr

EXPORT: DOWNSTREAM ADVECTION

Mass Flux 0.028 kg/hr

Percent of Load: 95.2 %

WATER COLUMN

Average Concentration: μg/l 0.029

Maximum Concentration: μg/l 0.029

Steady-state Accumulation: 99.8 %

Persistence[a]: 0 %

BIODEGRADATION BY WATER COLUMN BACTERIA

Mass Flux 0.00092 kg/hr

Percent of Load: 3.07%

BOTTOM SEDIMENTS

Average Concentration: μg/kg 0.026

Maximum Concentration: μg/kg 0.056

Steady-state Accumulation: 0.2 %

Persistence[a]: 83.4 %

BIODEGRADATION BY BOTTOM SEDIMENT BACTERIA

Mass Flux 0 kg/hr

Percent of Load: 0 %

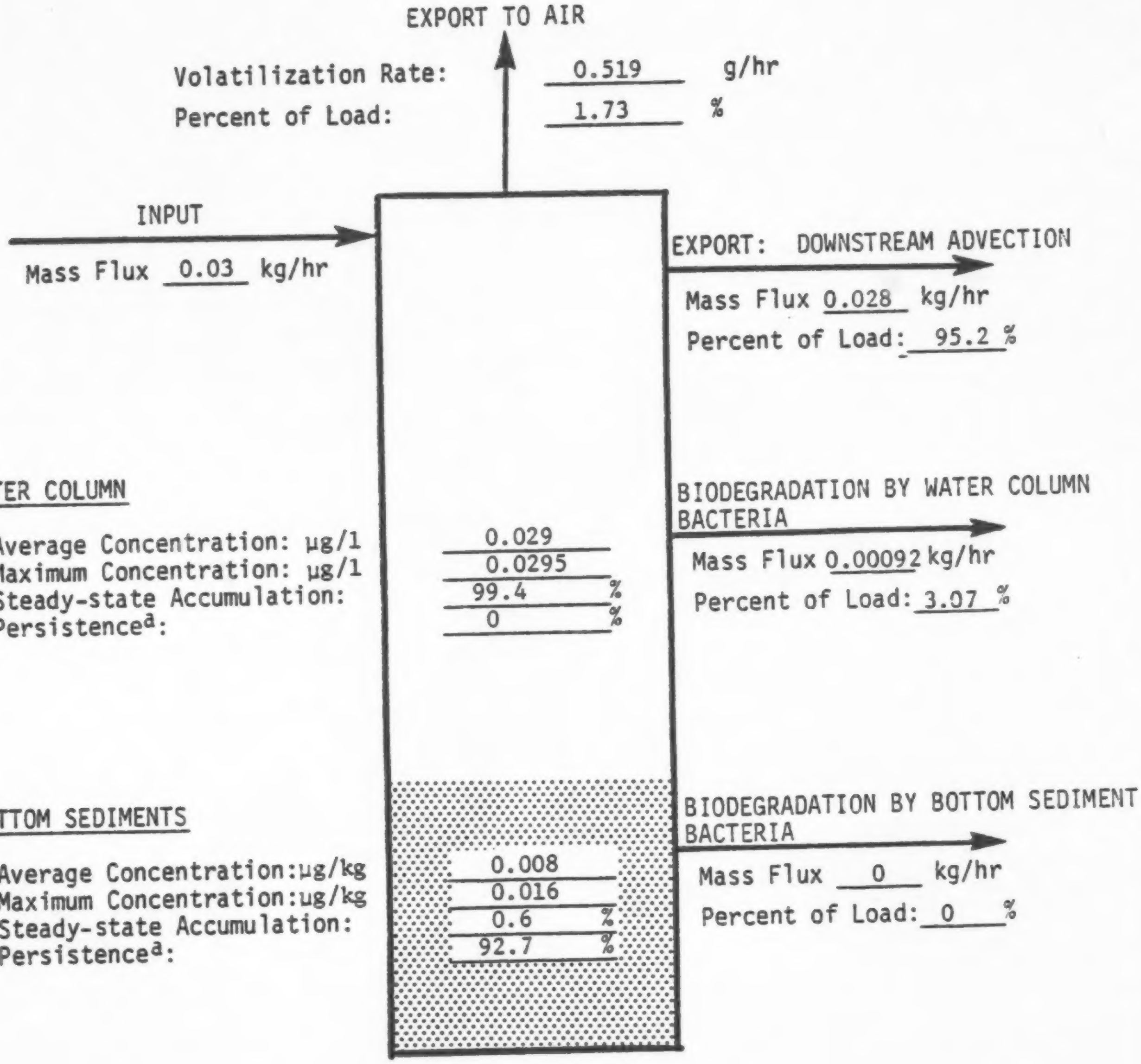
System Self Purification Time: 0.47 hour

Bioabsorbtion: Plankton: 6.6×10^{-4} μg/g; Bethos: 6.7×10^{-5} μg/g

[a] The percent of the pollutant remaining in that medium 12 hours after loading ceases.

FIGURE 4-5. RESULTS OF EXAMS MODELING OF THE ENVIRONMENTAL FATE OF BENZENE IN A CLEAN RIVER

Ecosystem: River: Benzene


EXPORT TO AIR
Volatilization Rate: 0.519 g/hr
Percent of Load: 1.73 %
INPUT
Mass Flux 0.03 kg/hr
EXPORT: DOWNSTREAM ADVECTION
Mass Flux 0.028 kg/hr
Percent of Load: 95.2 %
WATER COLUMN
Average Concentration: µg/l 0.029
Maximum Concentration: µg/l 0.0295
Steady-state Accumulation: 99.4 %
Persistence^a: 0 %
BIODEGRADATION BY WATER COLUMN BACTERIA
Mass Flux 0.00092 kg/hr
Percent of Load: 3.07 %
BOTTOM SEDIMENTS
Average Concentration: µg/kg 0.008
Maximum Concentration: ug/kg 0.016
Steady-state Accumulation: 0.6 %
Persistence^a: 92.7 %
BIODEGRADATION BY BOTTOM SEDIMENT BACTERIA
Mass Flux 0 kg/hr
Percent of Load: 0 %

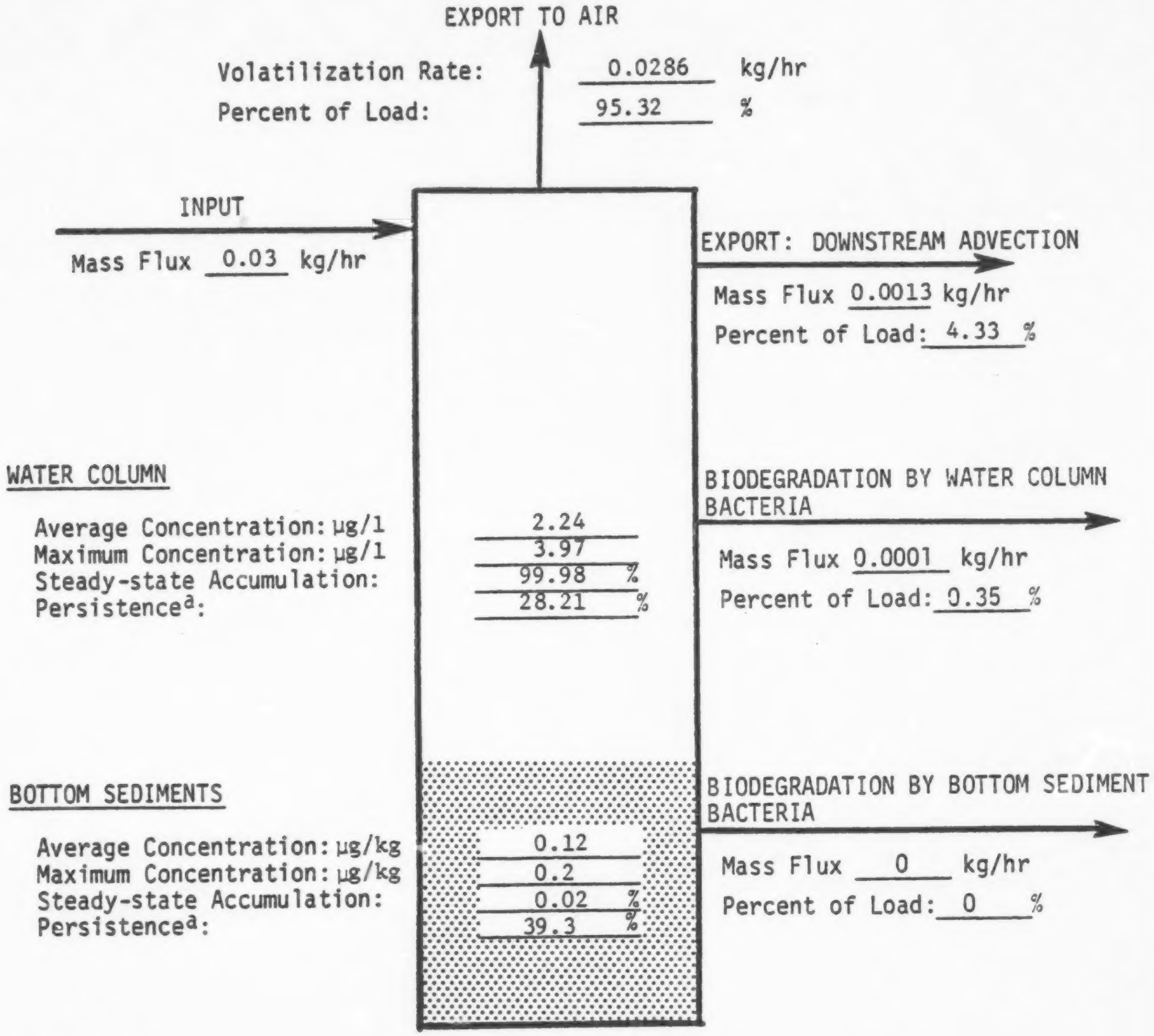
System Self Purification Time: 3.326 hour

Bioabsorbtion: Plankton: 6.6×10^{-4} µg/g; Benthos: 6.8×10^{-5} µg/g

[a] The percent of the pollutant remaining in that medium 12 hours after loading ceases.

FIGURE 4-6. RESULTS OF EXAMS MODELING OF THE ENVIRONMENTAL FATE OF BENZENE IN AN OLIGOTROPHIC LAKE

Ecosystem: Oligotrophic Lake: Benzene



EXPORT TO AIR

Volatilization Rate: 0.0286 kg/hr
Percent of Load: 95.32 %

INPUT

Mass Flux 0.03 kg/hr

EXPORT: DOWNSTREAM ADVECTION

Mass Flux 0.0013 kg/hr
Percent of Load: 4.33 %

WATER COLUMN

Average Concentration: μg/l 2.24
Maximum Concentration: μg/l 3.97
Steady-state Accumulation: 99.98 %
Persistence[a]: 28.21 %

BIODEGRADATION BY WATER COLUMN BACTERIA

Mass Flux 0.0001 kg/hr
Percent of Load: 0.35 %

BOTTOM SEDIMENTS

Average Concentration: μg/kg 0.12
Maximum Concentration: μg/kg 0.2
Steady-state Accumulation: 0.02 %
Persistence[a]: 39.3 %

BIODEGRADATION BY BOTTOM SEDIMENT BACTERIA

Mass Flux 0 kg/hr
Percent of Load: 0 %

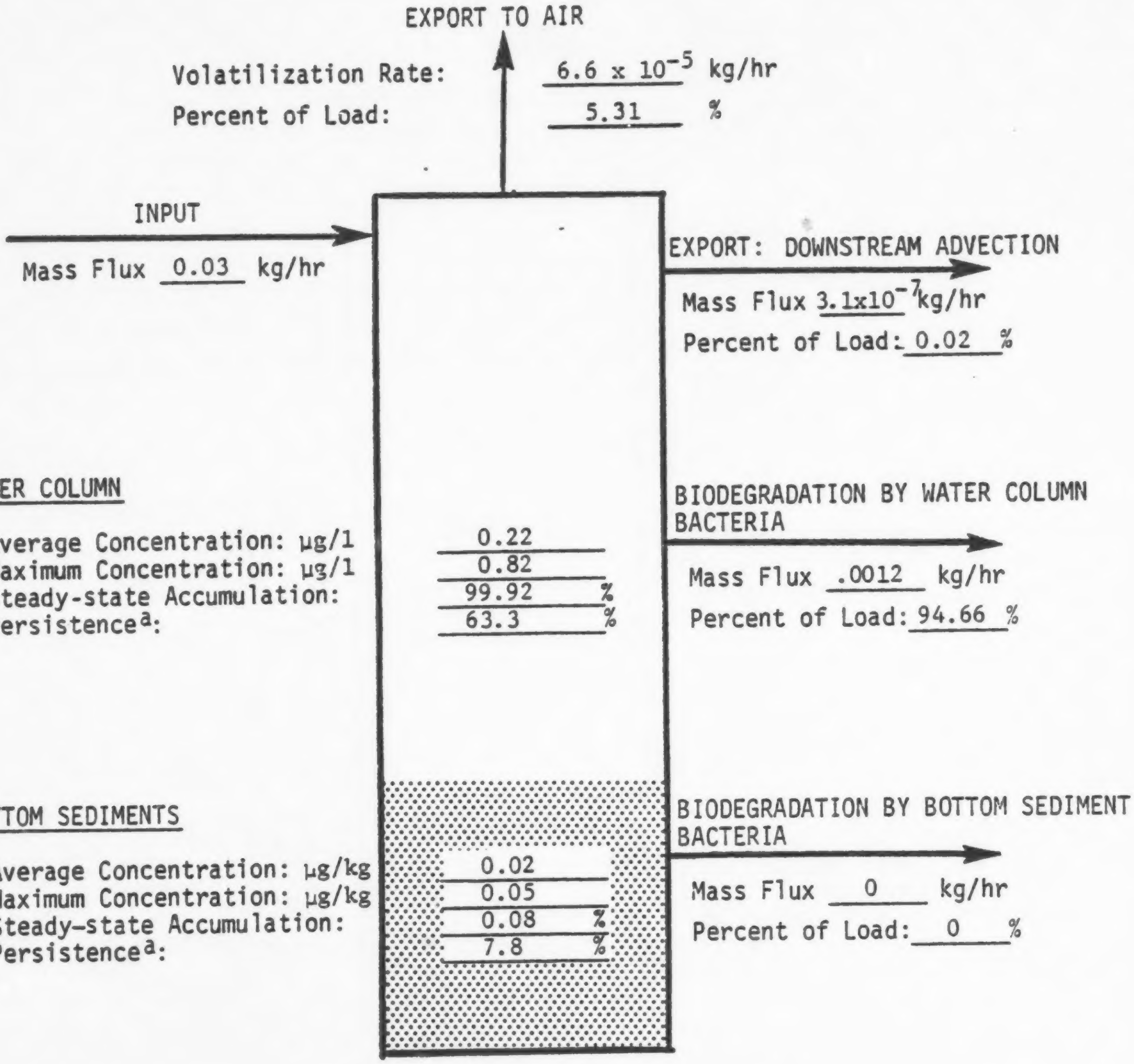
System Self Purification Time: 65.7 days

Bioabsorbtion: Plankton: 8.9×10^{-2} μg/g; Benthos: 1.3×10^{-3} μg/g

[a] The percent of the pollutant remaining in that medium 12 hours after loading ceases.

FIGURE 4-7. RESULTS OF EXAMS MODELING OF THE ENVIRONMENTAL FATE OF BENZENE IN A EUTROPHIC LAKE

Ecosystem: Eutrophic Lake: Benzene



EXPORT TO AIR

Volatilization Rate: 6.6×10^{-5} kg/hr

Percent of Load: 5.31 %

INPUT

Mass Flux 0.03 kg/hr

EXPORT: DOWNSTREAM ADVECTION

Mass Flux 3.1×10^{-7} kg/hr

Percent of Load: 0.02 %

WATER COLUMN

Average Concentration: µg/l 0.22

Maximum Concentration: µg/l 0.82

Steady-state Accumulation: 99.92 %

Persistence[a]: 63.3 %

BIODEGRADATION BY WATER COLUMN BACTERIA

Mass Flux .0012 kg/hr

Percent of Load: 94.66 %

BOTTOM SEDIMENTS

Average Concentration: µg/kg 0.02

Maximum Concentration: µg/kg 0.05

Steady-state Accumulation: 0.08 %

Persistence[a]: 7.8 %

BIODEGRADATION BY BOTTOM SEDIMENT BACTERIA

Mass Flux 0 kg/hr

Percent of Load: 0 %

System Self Purification Time: 41.9 hours

Bioabsorbtion: Plankton: 1.8×10^{-2} µg/g; Benthos: 3.6×10^{-4} µg/g

[a] The percent of the pollutant remaining in that medium 12 hours after loading ceases.

lake system, volatilization is also more important than export (5.31 vs. 0.02% of load). In this lake, however, EXAMS indicates that neither transport process is competitive with biodegradation (95% of load). This may be true if the bacteria concentration is high enough. Typically, one might expect to find the rate a factor of 10^2 to 10^3 slower than the 10^{-7} biodegradation rate estimate used in the basic EXAMS calculation. If the biolysis rate constant is on the order of 10^{-9}, then EXAMS would predict that 81% of the total benzene will volatilize and 15% will biodegrade in a eutrophic lake. In the river, removal will be determined primarily by environmental factors, with waterborne export and volatilization the dominant removal processes.

For each of the four ecosystems, the EXAMS-calculated partitioning between water and sediments is such that <99% of the benzene is in the water column and <1% in the bottom sediments.

The calculated half-lives for bacterial degradation volatilization and waterborne export for the four EXAMS scenarios are compared in Table 4-13. The biodegradation rates are unreasonably high when compared with the information presented in Sections 4.4.4.2 and 4.4.4.3 on biodegradation. A value of several days as opposed to 8-20 hours is more likely. Table 4-14 presents selected data from the EXAMS exposure analysis summary. The maximum calculated concentration in water for the assumed 0.03 g/hr loading is 4 μg/l in the oligotrophic lake; this is equivalent to about 27 days' total loading into the 5×10^6 m^3 lake volume. In all other aquatic compartments for all scenarios, the maximum calculated benzene concentration is <1 μg/l.

4.4.3 Intermedia Transfers

4.4.3.1 From Air Medium to Surface Waters or Land

Based on the materials balance analysis, air is the dominant receiving medium for benzene emissions. Furthermore, the vapor presure of benzene is sufficiently high (see Table 4-1) so that this chemical will have a strong tendency to remain in the atmosphere as a vapor. Dry deposition is not a plausible removal process for atmospheric benzene vapors because the vapor pressure of benzene is too high to attach to aerosols (Junge 1978).

Because the water solubility of benzene is relatively high, rainout as a removal mechanism may be measurable. Still, the amount of benzene removed by this process is small.

To estimate the potential importance of rainout as an intermedia transfer mechanism, it is useful to:

- determine the equilibrium partitioning between rain and atmosphere; and
- consider a local scenario, with its annual rainfall, benzene concentration in the air and rainwater to calculate percent rainout of benzene as an example.

TABLE 4-13. HALF-LIVES FOR TRANSFORMATION AND TRANSPORT OF BENZENE FOR SEVERAL EXAMS SCENARIOS

	Half-life (hr)			
	River		Lake	
	Clean	Turbid	Oligotrophic	Eutrophic
Bacterial Degradation				
Water column	20	20	66,000	7.9
Bottom sediments	280	91	109,000	140
Volatilization	35	35	240	140
Waterborne	0.6	0.6	5,400	30,000
Total Transformation and Transport	0.6	0.6	230	7.5

Source: Arthur D. Little, Inc.

TABLE 4-14. EXPOSURE ANALYSIS SUMMARY FOR BENZENE FROM EXAMS MODEL RUNS

	Benzene 0.03 kg/hr Input Loading			
	River		Lake	
	Clean	Turbid	Oligotrophic	Eutrophic
Maximum Concentrations				
in Water Column, g/l	0.03	0.03	4	0.8
in Plankton, g/l	0.0007	0.0007	.09	0.02
in Benthic Organisms, g/g	0.00007	0.00007	.001	0.0004
in Bottom Sediments, g/kg	0.016	0.006	0.21	0.05
Total Steady State Accumulation in Aquatic Ecosystem				
kg	0.026	0.026	10	0.32
% in Water Column	99.39	99.80	99.98	99.92
% in Bottom Sediments	0.61	0.20	0.02	0.08
Persistence				
Recovery Period, hr[a]	12	12	576	12
% of Initial Benzene Burden Lost from:				
Water Column	100	100	72	63
Bottom Sediments	7.3	17	61	7.8

[a] Time after loading ceases. This parameter is based on EXAM's definition of persistance. An alternative explanation is given by the downstream distance required for concentrations to approach zero (see p. 4-33).

Source: Arthur D. Little, Inc.

These calculations do not determine a rate for benzene rainout, rather, they determine the percentage of benzene in the atmosphere that could reach the surface as a result of rainout during a specific rainfall event. Thus, monitoring data for atmospheric benzene at a given location were used along with rainfall data for that same location. Although the concentration of benzene in the atmosphere as well as the amount of rainfall in each event may fluctuate, this approach will indicate the significance of rainout as a removal mechanism.

No data are available on the concentration of benzene in rain, so the equilibrium partitioning cannot be determined from actual concentration data. However, the concentration of benzene in the rain can be estimated if the concentration in the air is known and if the Henry's Law constant is either known or calculable. Unless the benzene contaminated air is confined to a low altitude, the droplets and the contaminated air can be expected to reach equilibrium. In the case of a confined "dirty" air mass, raindrops falling through the mass would have insufficient time to reach equilibrium; thus, they could not contribute to rainout. Assuming that equilibrium may be attained, the concentration of benzene in rain can be estimated by the following expression:

$$C_{air} = HC_{rain} \qquad \text{Eq. 4.4-1}$$

where H is the Henry's Law constant, C_{air} is the concentration of benzene in the air, and C_{rain} is the concentration of benzene in rain. H may be written in a nondimensional form:

$$H = \left(\frac{P_r}{P_t}\right)\left(\frac{M}{29}\right)\left(\frac{P_{air}}{X_s}\right) = 0.24 \text{ for benzene} \qquad \text{Eq. 4.4-2}$$

where: P_r = vapor pressure of benzene = 0.125 atm

P_t = partial pressure of air = 1 atm

M = molecular weight of benzene = 78.1 (g/mole)

29 = "molecular weight" of air (g/mole)

P_{air} = density of air 1.29 g/l (1.29 kg/m^3)

X_s = solubility of benzene = 1.78 g/l

Using this estimate of the Henry's Law constant, the rainfall concentration of benzene can be estimated for any given air concentration of benzene.

Riverside, California was selected for this example. Because of benzene producing petroleum plants nearby in El Segundo and a high volume of vehicles on the road in this portion of California (see Chapter 3.0), a detectable concentration of benzene will exist in the air. Monitoring data have shown ambient air concentrations of benzene of 25.5 $\mu g/m^3$. Using Equation 4.4-1, the corresponding rain concentration of benzene is calculated to be 0.1 $\mu g/l$. Assuming an annual precipitation in Riverside of 0.51 m with an annual average of 65 rainfall events (days), then the average rainfall event is 7.85×10^{-3} m. Using the above variables, the quantity of benzene lost from the atmosphere via rain can be calculated.

Let T_r = quantity of benzene lost from the atmosphere ($\mu g/m^2$)

C_{rain} = concentration of benzene in the rain ($\mu g/m^3$)

M_{pa} = quantity of average rainfall event (m)

such that $T_r = C_{rain} M_{pa}$ Eq. 4.4-3

For this example, $T_r = 0.83\ \mu g/m^2$. To place rainout in perspective, the percentage of atmospheric benzene released during rainout was determined.

Monitoring data show the benzene concentration to be 25.5 $\mu g/m^3$ in air. If the mixing depth is 1 kilometer, then the amount of benzene in the air is about $25.5 \times 10^3\ \mu g/m^2$. Rainout decreases the amount of benzene in the air by 0.83 $\mu g/m^2$, which is much less than 1% of the total. Therefore, the role of rainout in reducing atmospheric benzene is slight. Most atmospheric benzene remains in the air where its ultimate fate is determined by intramedia processes.

4.4.3.2 Intermedia Transfers from Water Medium

Water to Air

Volatilization is an important process in the depletion of benzene from water. Benzene emitted to the water compartment either by direct entry or by chemical process is decreased because of benzene's high volatility.

The half-life for volatilization of benzene in water depends on both physical and chemical parameters. Physical parameters describe the physical properties of the given scenario, such as the depth of the water body (D), the wind speed (V_w), and the current speed (V_c). The chemical parameters are the liquid-phase exchange coefficient (k_l), the gas phase exchange coefficient (k_g), the liquid phase mass transfer (K_L), the molecular weight (M), and the nondimensional Henry's Law constant (H).

If values are assumed for the physical parameters such as,

$V_w = 2$ m/s

$V_c = 1$ m/s

$D = 1$ m

(values for V_w are from Battelle 1979, V is from Mackay and Leinonen 1975, V_c is assumed) then the chemical parameters can be estimated using the following equations from Southworth (1979);

$$k_1 = 23.51 \left(\frac{V_c^{0.969}}{D^{0.673}}\right) \sqrt{32/M} \quad e^{0.526\,(V_w - 1.9)} = 16 \text{ cm/hr}$$

$$kg = 1137.5\,(V_w + V_c) \quad \sqrt{18/M} = 1639 \text{ cm/hr}$$

$$k_L = \frac{H\,k_g\,k_\ell}{H\,k_g\,k_\ell} = 15 \text{ cm/hr.}$$

Using the value of the liquid phase mass transfers, k_L, the half-life is estimated to be:

$$t_{1/2} = 0.693\ D/k_L = 4.6 \text{ hr.}$$

Given that the half-life is 4.6 hours and the current velocity, V_c, is 1 m/s, then the distance downstream the water would flow before 50% of the benzene had volatilized would be:

$$\text{Distance Downstream} = t_{1/2}\ (\text{hr}) \times V_c(\text{m/s}) \times 3600\ (\text{s/hr})$$

$$= 16{,}560 \text{ m.}$$

In addition to volatilization, benzene in the water segment will be diffused throughout the water column and will be adsorbed by the sediment and aquatic organisms. The EXAMS model accounts for the benzene that will be both volatilized and diffused. Therefore, from the output of the EXAMS model, downstream distances can be calculated that estimate the distance the benzene in the water segment would flow until some percentage of the load has either evaporated or been adsorbed by sediments and organisms.

For all the scenarios used in the EXAMS model, the current velocity is 0.93 m/sec, the depth of the water column is 3 meters, the width and length of the water column are 100 and 1000 meters, respectively, and the water flow rate is 2.41×10^7 m^3/day. In both turbid and clean rivers, 95.2% of the benzene in the original flux into the river segment analyzed is passed onto the next segment, 1.73% volatilizes, and 3.07% is biodegraded. The physical representation of an EXAMS river allows the use of an exponential decay function to solve for the number of river segments through which the benzene load must flow until some percentage (i.e., 99%) of the load either leaves the water compartment by evaporation or by adsorption by sediments and organisms.

The calculation is:

$$(\text{mass flux \% to next river segment})^n = 0.01$$

where n is the number of segments necessary to obtain a 99% reduction in the initial loading of benzene to the river. Solving:

$$n \log (0.952) = \log 0.01$$
$$n = 93.62.$$

Because each EXAMS river segment is 1000 meters long, this distance is 93,620 meters (58.17 miles); with a water velocity of 0.93 m/sec, only 1.16 days is necessary for this 99% reduction to occur. Similar calculations show that a reduction of 50% of the initial load occurs in 4.2 hours over a river stretch of 14,090 meters (8.76 miles). Figure 4-8 shows distances downstream at various percentages of benzene reduction.

Water to Soil

As shown by the EXAMS model, benzene transfers from water to the sediment with 3.07% of the benzene binding to the sediment and aquatic organics. In addition, the log octanol-water partition coefficient (2.13) shows that benzene does have an affinity to bind to the organics in soil. However, the extent to which benzene is transferred depends on the sorption capability of the particular soil which in turn depends on the organic content and the porosity. (See the section on soil adsorption for additional information on this process.)

4.4.3.3 Intermedia Transfers from Soil Medium

The materials balance analysis indicates that approximately 228 kkg/yr of benzene is introduced into the soil medium by land disposal of wastes from petroleum refining. Depending on whether this is surface land disposal (e.g., lagooning or land farming) or subsurface land disposal (e.g., landfilling), the benzene may migrate to the air or to water.

Soil to Water

The magnitude of the soil:water partition coefficient for benzene suggests that transport of benzene from the soil medium to ground water via leaching or to surface water via runoff may be an important environmental fate process.

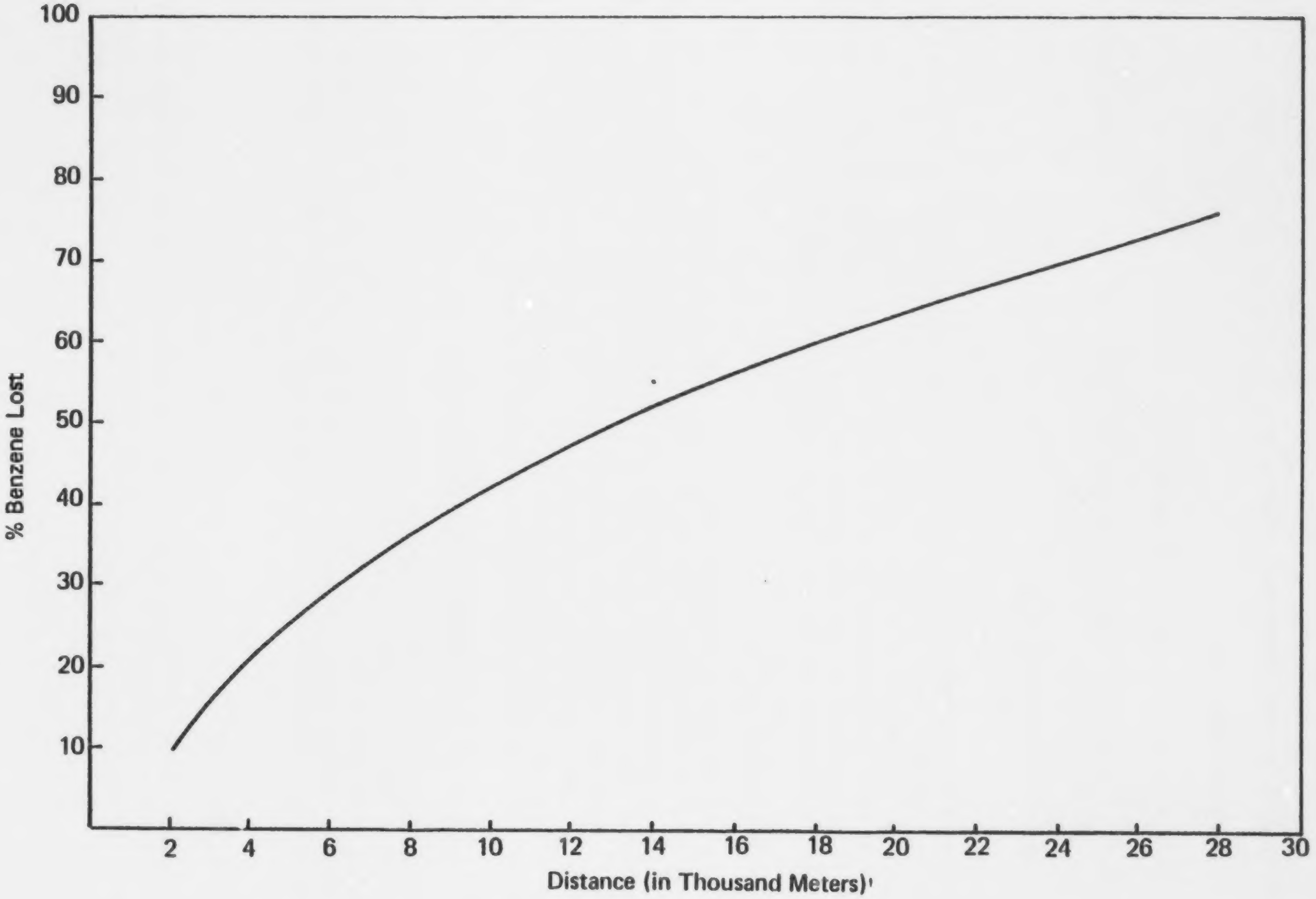


FIGURE 4–8 PERCENTAGE OF BENZENE REDUCTION AT DOWNSTREAM DISTANCES FROM AQUATIC DISCHARGES IN A 3-METER DEEP STREAM

The partition coefficient is given by:

$$K_{oc} = \left(\frac{\text{C benzene in soil}}{\text{C benzene in water}}\right) X \left(\frac{1}{\text{fraction organic carbon in soil}}\right)$$

and $K_{oc} = 74$ when the benzene concentrations are given in µg/kg of soil and µg/l of water. If 1 kg of soil with 1% organic carbon content were equilibrated with one of water, the concentration of benzene and the actual benzene mass loading would be approximately 1.3 times higher in the aqueous phase than in the soil, based on this value of K_{oc}. Transfer of some soil-associated benzene to water is a real possibility as shown by these simple calculations, (but probably not a dominant fate mechanism as shown by equilibrium partitioning).

Estimation of the probable quantitative significance of soil-to water transport of benzene, however, is not generally feasible for two reasons. First, it is not possible to specify the volume of water (runoff or leachate) that could realistically be assumed to contact any given quantity of soil in the environment. Thus, the equilibrium mass loading of benzene in the two media cannot be calculated, even though the ratios of concentrations could be estimated from K_{oc}. Second, this intermedia transfer in the environment involves transport across the solid-liquid phase interface and is affected by kinetic as well as equilibrium phenomena. The K_{oc} calculation is an indication of equilibrium steady state; however, it provides no information on the possible rate of transfer process.

Monitoring data on benzene levels in actual or simulated leachate/runoff situations would be helpful in developing estimates of the significance of soil-to-water transport of this chemical. However, few data on soil levels and no data on leachate/runoff were found.

In laboratory research by Rogers et al. (1980) on various soils, it was concluded , "Although sorbed benzene tends to resist desorption, a miminal amount of benzene sorbed to solid would be lost to water." They found soil benzene to be "tightly bound" in general, although their work covers essentially only two soil types.

Soil to Air

The physical chemical properties of benzene indicate that volatilization is the most probable transfer pathway for loss of benzene from the soil surface. The equilibrium constant for this process, K_{vol}, can be estimated from the Henry's Law constant and the soil adsorption constant as follows:

$$\frac{C_{air}}{C_{water}} = H$$

$$\frac{C_{soil}}{C_{water}} = K_{oc} \text{ X fraction o.c. in soil}$$

$$\frac{C_{air}}{C_{soil}} = \frac{H}{K_{oc} \text{ X fraction o.c. in soil}} = K_{vol.}$$

For benzene, assuming a typical soil organic carbon content of 1%, an H value of 229 (see Table 4-12) and a K_{oc} value of 74 (see Table 4-1) K_{vol} is numerically equal to:

$$K_{vol} = \frac{229}{74 \text{ X } 0.01} = 310 \ \frac{\text{mg/m}^3 \text{ in air}}{\text{mg/kg in soil.}}$$

This indicates a strong driving force for volatilization of benzene from surface soil.

A soil concentration of 25 mg/kg in soil, which is within the range observed in the vicinity of chemical plants (see Section 3.2), corresponds to a calculated equilibrium air concentration of 7800 mg/m^3 or about 8 g/m^3. This value is considerably higher than the 1-100 µg/m^3 benzene concentrations reported for air samples in the vicinity of chemical plants (see Table 4-3). It is, however, greater than the maximum air level recorded above a landfill site of 900 mg/m^3. Because the levels of benzene within the wastes or soils at this landfill were not documented, the 900 mg/m^3 may be representative of the local equilibrium conditions. Downwind levels from this site were between 0.01 and 1.55 mg/m^3 demonstrating dispersion effects while upwind levels (possibly indicative of diffusion effects) were measured at trace to 0.2 mg/m^3. These results are consistent with the expectation that "equilibrium" concentrations would not be typical except in a shallow layer of air near the soil surface. Dispersion and diffusion would rapidly reduce benzene concentrations by several orders of magnitude.

4.4.4 Intramedia Fate Processes

4.4.4.1 Air

The predominant fate process within the air medium are dispersion and reaction with hydroxyl radical.

Dispersion

A model for the atmospheric dispersion of benzene by Youngblood of the U.S. EPA has been reported in a recent U.S. EPA document on human exposure to benzene (Mara and Lee 1978). Youngblood's modeling effort was based on D. B. Turner's (1970) modeling techniques from which ground-level and 1-hour worst case concentrations were derived. Eight-hour worst-case concentrations were also estimated by multiplying the one-hour concentrations by 0.5. Concentration estimates at distances up to 20 kilometers from the source were derived by setting a fixed emission rate (100 g/s) and by assuming a 4 m/s wind speed and a neutral stability class. Excerpts of the results of Youngblood's analysis follow.

Chemical Manufacturing Plants

In 1978, chemical plants used more than 5 million kkg of benzene as an intermediate in the production of chemicals. To assess the impact of benzene consumption in the chemical manufacturing industry on benzene concentrations in the atmosphere, a point-source model (Turner 1970) was used. Because the height of the source point varies among plants, three representative heights were chosen. The three emission source heights were: A, ground-level (effective stack height = 0 m), B, building level (effective stack height = 10 m), and C, elevated source level (effective stack height = 20 m). For calculations involving the building height source, the results from Turner's workbook (1970) were adjusted to account for the initial dispersion of benzene in the building cavity. A single dispersion curve was subsequently developed to represent all three source height categories by averaging the high and low value of the three emission source categories at each distance. Table 4-15 and Figure 4-9 show the 8-hr worst case ground-level concentration of benzene around a chemical manufacturing plant among the three source heights. Benzene concentrations vary greatly closest to the source of emission; however, they taper off quickly so that at 0.3 km the concentrations from all three hypothetical source heights are within the uncertainty level of the dispersion calculation. Distances <0.4 km are likely to be within the plant perimeter or at least to have low population densities (Mara and Lee 1978). Therefore, the concentration of benzene outside the plant perimeter does not depend on the height of the source. Extrapolating from the "M curve" in Figure 4-9, the 8-hr worst case benzene concentration is roughly 5000 $\mu g/m^3$ at 0.4 km. The 1-hr worst case concentration would be twice as great while the annual average would be 200 $\mu g/m^3$. Monitoring data from five chemical plants show concentrations of benzene within 1 km of the plant to range from 1.6 to 186.4 $\mu g/m^3$ (RTI 1977). The annual average concentration, using Turner's point source dispersion model, falls within 10% of the concentration actually observed.

Coke Ovens and Petroleum Refineries

Atmospheric dispersion modeling of benzene from coke-oven operations and petroleum refineries requires a model that accounts for the large area of the emissions source. Unlike the chemical plant example above, emissions from coke ovens and petroleum refineries are not restricted to a single point source, but rather emanate from several sources during the coal to coke conversion processes and petroleum refining processes. To account for the large area of release, Youngblood (1977) used the Point, Area, and Line Source (PAL) Dispersion Model (Turner and Peterson 1975). For coke ovens operations, the assumptions used for this model inlcude:

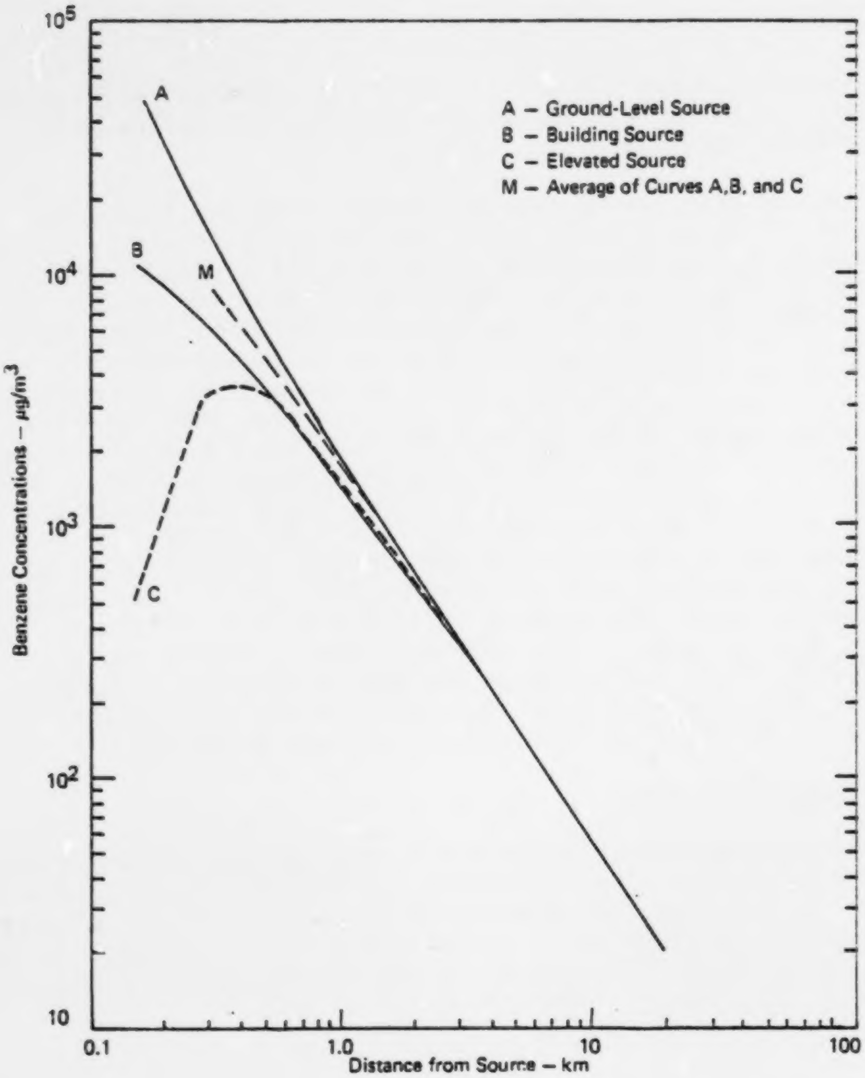
- Benzene emissions occur mostly from oven leaks,
- The plant site is square,

TABLE 4-15. ROUGH ESTIMATES OF AMBIENT GROUND-LEVEL BENZENE CONCENTRATIONS (8-HOUR) AVERAGE[a] PER 100 g/s EMISSION RATE FROM A CHEMICAL MANUFACTURING PLANT

Source Category	Benzene ($\mu g/m^3$)											
	0.15 km	0.3 km	0.45 km	0.6 km	0.75 km	1.6 km	2.5 km	4.0 km	6.0 km	9.0 km	14.0 km	20.0 km
A	51,000	14,000	7,000	4,500	3,000	900	440	220	120	62	34	20
B	11,000	6,100	3,800	2,800	2,100	740	370	220	120	62	34	20
C	510	3,500	3,500	2,800	2,100	800	410	220	120	62	34	20

[a] To give rough estimates of annual average concentrations, multiply by 0.04.

Source: Youngblood (1977).



Source: Youngblood (1977).

[a] Based on an emission rate of 100 grams

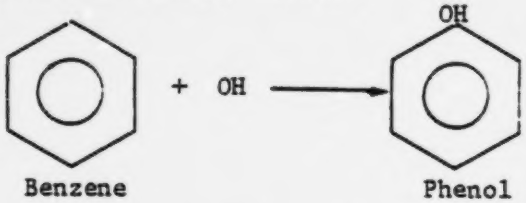
FIGURE 4–9 DISPERSION MODELING RESULTS FOR EACH TYPE OF SOURCE CATEGORY[a]

- The emissions are uniformly distributed throughout the specified area,
- Effective stack height = 10 meters,
- Wind speed = 4 m/s,
- Stability class = neutral, and
- Emissions rate = 100 g/s. (This is an unusually high rate. Most coke-oven operations and petroleum refineries have emission rates less than 10 g/s.)

The concentration of benzene at gradual distances from plants of varying areas is shown in Table 4-16. For the smallest plant, the concentration ranges from 20 $\mu g/m^3$ at 20 km from the source to 5000 $\mu g/m^3$ at only 0.3 km from the source. The larger plants show smaller concentrations at corresponding distances outside their boundaries, which would be expected because the plant size has increased while the emission rate has remained constant. Note that the benzene concentrations from the smallest plant (0.01 km^2) at each distance from the source fall within the range of the concentrations of benzene at the corresponding distances from the chemical manufacturing plant. For a coke oven or petroleum refineries facility with a larger area (Mara and Lee 1978 choose 0.25 km^2 as a "typical plant area" for a coke-oven plant), the concentration is much less than the 0.01 km^2 plant. Furthermore, the emission rate used in this calculation (100 g/s) is significantly greater than the usual coke-oven or petroleum refineries emission rate (10 g/s) of Mara and Lee (1978). The anticipated benzene concentration in the vicinity of these facilities can then be expected to be much smaller than those reported here and, consequently, much smaller than those found in the vicinity of a chemical manufacturing plant.

Atmospheric Oxidation

The rate of benzene depletion due to free radical oxidation directly depends on the reaction rate of benzene with hydroxyl radicals and with ozone. The reaction rate of benzene with hydroxyl radicals is considerably faster than the reaction rate with ozone, and, therefore, is the rate limiting reaction. The product of the benzene/hydroxyl radical reaction is phenol (Hendry 1978).



The rate of benzene depletion due to free radical oxidation also depends on the concentration of hydroxyl radicals and ozone in the atmosphere. Table 4-17 illustrates that the concentration varies depending on the environmental setting. Rate constants for the benzene-hydroxyl radical reaction, k_{OH}, and the benzene-ozone reaction, k_{O_3}, have been estimated to be 8.4×10^{11} cm^3/mole-sec and 28 cm^3/mole-sec, respectively (Hendry 1978).

TABLE 4-16. ROUGH ESTIMATES OF 8-HOUR WORST CASE BENZENE CONCENTRATIONS PER 100 g/s EMISSION RATE USING THE PAL DISPERSION MODEL FOR A CHEMICAL MANUFACTURING PLANT

Distance From Source Area (km)	Benzene ($\mu g/m^3$) for Given Plant Area						
	0.01 km^2	.06 km^2	0.25 km^2	1 km^2	4 km^2	9 km^2	25 km^2
0.3	5,000	2,000	900	365	145	80	39
0.45	3,850	1,700	750	325	130	75	37
0.60	2,850	1,450	650	290	120	70	34
0.75	2,150	1,250	595	260	110	65	33
1.6	800	600	390	190	85	50	27
2.5	405	360	270	150	70	43	23
4.0	205	190	165	110	50	35	20
6.0	110	110	100	80	45	29	17
9.0	60	60	55	50	34	23	14
14.0	33	32	32	29	23	18	11
20.0	20	20	19	18	16	13	9

Source: Youngblood (1977).

TABLE 4-17. CONCENTRATIONS OF HYDROXYL RADICALS AND OZONE IN ATMOSPHERE OF DIFFERENT ENVIRONMENTAL SETTINGS

	Concentration of OH (mole/cm^3)	Concentration of O_3 (mole/cm^3)
Urban	5×10^{-18}	1×10^{-12}
Rural	5×10^{-19}	1.6×10^{-12}

Source: Arthur D. Little, Inc.

TABLE 4-18. OXIDATION RATE CONSTANTS AND HALF-LIVES OF BENZENE IN DIFFERENT ENVIRONMENTAL SETTINGS

	Urban	Rural
k_{ox} (sec^{-1})	4.2×10^{-6}	4.2×10^{-7}
$t_{1/2}$ (hrs)	46	458

Source: Arthur D. Little, Inc.

From these rate constants, the overall oxidation rate, k_{ox} can be calculated using the following equation:

$$k_{ox} = k_{OH}[OH] + k_{O_3}\,[O_3]$$

where k_{ox} = rate of oxidation (sec^{-1})

K_{OH} = rate constant of benzene with hydroxyl radical $\left(\frac{cm^3}{mole\text{-}sec}\right)$

$[OH]$ = concentration of OH radical in troposphere $\left(\frac{mole}{cm^3}\right)$

$[O_3]$ = concentration of O_3 radical in troposphere $\left(\frac{mole}{cm^3}\right)$

k_{O_3} = rate constant of benzene with ozone $\left(\frac{cm^3}{mole\text{-}sec}\right)$

For an urban environment, the atmosphere oxidation rate of benzene is 4.2×10^{-6} sec^{-1} while for a rural environment, the rate of atmospheric oxidation is slighter lower 4.2×10^{-7} sec^{-1}, because of the slightly smaller hydroxyl radical concentration. Knowing the oxidation reaction rate constant, k_{ox}, the half-life of benzene in the atmosphere can be calculated using the following expression:

$$t_{1/2} = \frac{0.693}{k_{ox}}$$

The half-lives of benzene in the atmosphere are shown in Table 4-18. Similar half-lives were calculated by Davis et al. (1977) who evaluated the concentration of hydroxyl radicals over a range of latitudes for different hours of the day during both winter and summer months. The reaction of benzene with ozone was not addressed in their work because the rate of reaction is so slow that it has minimal effect on the rate of oxidation. Table 4-19 lists the hydroxyl radical concentration (measured here in mole/cm^3) and the half-lives of benzene calculated on the basis of $k_{OH} = 1.59 \times 10^{-12}$ cm^3/mole-sec. The average half-life of benzene in the lower atmosphere ranges from 12 to 121 hours, which is in the same range as the half-life observed using an average hydroxyl radical concentration. Therefore, using a location and time specific concentration or an average concentration yields the same result -- a half-life of benzene in the atmosphere of a few days.

Since benzene undergoes atmospheric oxidation between 2 and 20 days and does not in any other way react chemically (such as via photolysis), it is expected that transport mechanisms will become a more significant factor in the dispersion of benzene than chemical mechanisms. Thus, atmospheric dispersion can be expected to carry benzene a great distance from the source of emissions, making benzene more of a regional problem and less of a localized atmospheric problem.

Photochemistry

Howard and Durkin (1974), Walker (1976) and Mara and Lee (1978) note previous reviews on the photochemistry of benzene. Benzene has

TABLE 4-19. HALF-LIFE OF BENZENE IN THE LOWER TROPOSPHERE[a]

	Latitude, Time of Day, and Season for Various Model Conditions							
	30°N 8 AM Summer	30°N 12 Noon Summer	30°N Diurnal Summer	30°N 12 Noon Winter	30°N Diurnal Winter	70°N 12 Noon Summer	70°N Diurnal Summer	37°N 12 Noon Summer
Concentration of OH (in millions of moles/cm^3)	6	10	4	2	1	3	2	9
$T_{1/2}$ (hr)	20	12	30	61	121	40	61	13

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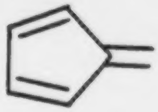
[a] $K_{OH} = 1.59 \times 10^{-12}$ cm^3/mole-sec

$$t_{1/2} = \frac{.693}{K_{O_x}}$$

Source: Davis _et al_. (1977).

essentially no absorbance at wavelengths >290 nm; consequently, it will be nonreactive by direct photochemical processes under normal conditions in the troposphere or on the earth's surface. This view is consistent with the observation of Altshuller and Bellar (1971) that benzene is "essentially unreactive" in photochemical smog chamber studies.

Several laboratory studies of the photochemistry of benzene at wavelengths <290 nm have been reported. On gas phase irradiation with UV light ($\lambda = 254$ nm), benzene has been reported (Bryce-Smith and Gilbert 1976) to form rearrangement products, such as fulvene (below) with quantum



yields on the order of 0.01-0.03. Higher energy UV light (147 nm) has been reported (Jackson et al. 1967) to lead to the formation of low molecular gaseous decomposition products of benzene: acetylene, ethylene, butene -- in low yield, along with polymeric decomposition products. Noyes et al. (1966) reveal that fluorescent emission from the triplet state is one of the major decay paths (quantum yield about 0.22) for photochemically excited benzene. Fluorescence and radiationless decay to the ground state thus appear to be dominant processes for benzene excited states, even when high energy ($\lambda <254$ nm) radiation is employed. Neither process results in depletion of benzene from the gas phase.

4.4.4.2 Water

Benzene is inert with regard to the chemical transformation processes-- hydrolysis, photolysis, oxidation-- that typically occur within aquatic environments. Its aromatic ring creates a negative charge-density that impedes nucleophilic attack (Morrison and Boyd 1973). Diffusion is an important physical intramedium fate process for benzene because it leads to a major intermedia pathway: volatilization from water to air. Biodegradation of benzene appears to be an important fate process. In wastewater treatment plants (POTW's), efficient breakdown of aromatic organics is of concern for water quality; and because of benzene's solubility, biodegradation in natural waters could be important.

Biodegradation

Although no microorganism is known that can directly cleave the unsubstituted benzene ring, benzene-degrading populations (of unidentified species) or species have been isolated from activated sludge, domestic sewage, petroleum waste, and marine and river water and sediment (Howard and Durkin 1974). This indicates a widespread distribution of potential benzene degraders. Almost all benzene degraders were isolated from different habitats because of their ability to grow on benzene, toluene or aromatics in general. These organisms were subsequently exposed to benzene alone.

Degradation Pathways

Two major pathways are commonly followed in the microbial degradation of benzene (Swisher 1970); the first step in both reactions is oxidation to catechol and then splitting the ring either between or adjacent to the two hydroxyl groups. The two pathways and their metabolic products are depicted in Figure 4-10. A third pathway has been reported for phenol-acclimated sludge. Both reactions produce compounds that are commonly found in cell metabolites or components.

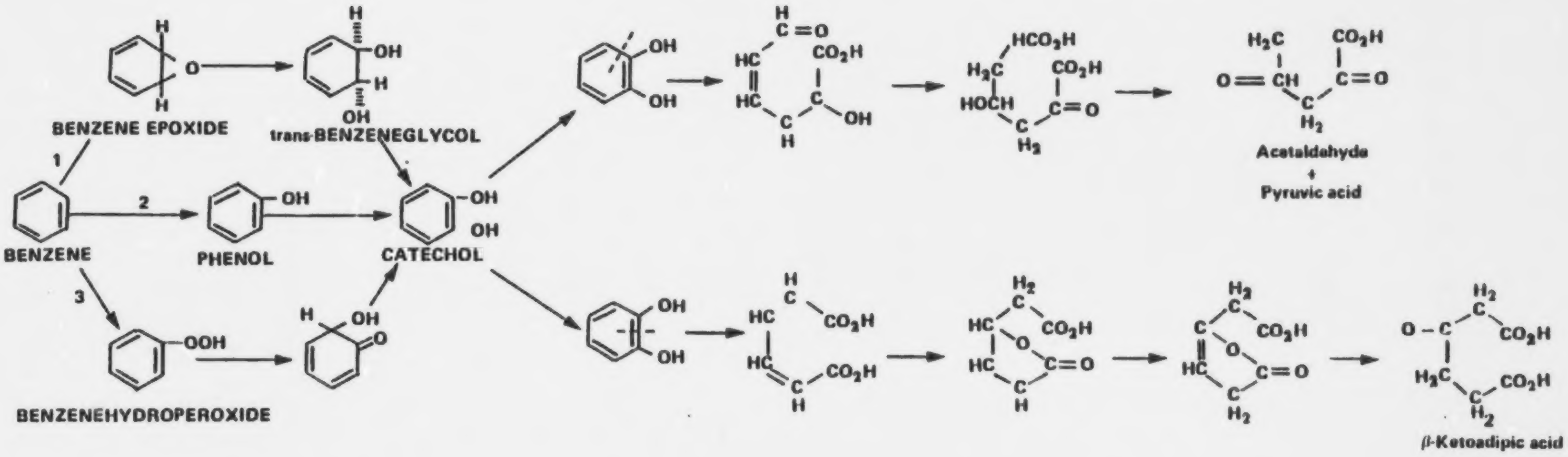
Several hypotheses have been suggested concerning the first step of the reaction between benzene and catechol (Gibson et al. 1968). The first theory (see 1, Figure 4-10) is that the benzene nucleus undergoes expoxidation, then hydrolysis to produce transbenzene glycol, which is dehydrogenated to catechol (Taniuchi et al. 1964). A second hypothesis is that benzene goes through a monohydroxylation reaction to phenol, then hydroxylation to catechol (see 2, Figure 4-10). The third is that a hydroperoxide is formed and undergoes hydroxylation to catechol (see 3, Figure 4-10). The third hypothesis has been tested using *Pseudomonas putida* and the results, though tentative, are supportive (Gibson et al. 1968, 1970). Because the first step is probably ratelimiting (Marr and Stone 1961), it should be examined, if it has not been, for a variety of microbial species.

Degradation Rates

It is difficult to compare the results of different experiments because biodegradation tests, in general, are variable and observations are rarely quantified as rate constant. Consequently, it is impossible to estimate a benzene biodegradation rate and the extent to which controlling variables influence it. Using the oxygen consumption measurements presented in Table 4-20, however, an examination of the rates measured will give an idea of the variability in rates. Slower rates may be due to the lack of acclimation and/or a short experimental period. Other factors that may have influenced the rates include the presence of alternative carbon sources or oxygen consumption by activities not involving benzene (e.g., endogenous respiration). Extrapolating these results to environmental conditions is difficult; however, in most cases, controlled laboratory conditions are more conducive to degradation (using adapted populations, providing optimum temperature and benzene concentration, controlling volatilization) than are natural conditions. Consequently, the results reported in Table 4-20 can generally be considered as upper limit rates. Reported rates ranged from 45% in 10 hours to 0.6% in 1 week.

Biodegradation During Wastewater Treatment

Biodegradation of benzene appears to occur during wastewater treatment. In a survey of the susceptibility of numerous substances to biological wastewater treatment, Thom and Ag (1975) classified benzene as biodegradable following acclimation. In contrast, Helfgott et al.


BENZENE EPOXIDE
trans-BENZENEGLYCOL
1
2
3
BENZENE
PHENOL
CATECHOL
BENZENEHYDROPEROXIDE
Acetaldehyde
+
Pyruvic acid
β-Ketoadipic acid

Source: Gibson et al. (1968), Howard and Durkin (1974).

FIGURE 4-10 DEGRADATION PATHWAYS FOR BENZENE

TABLE 4-20. BENZENE BIODEGRADATION RATES[a]

% Degraded	Time	Reference
3.2	6 hr; 5 days	Bogan and Sawyer (1955)
45[b]	10 hr	Okey and Bogan (1965)
33	12 hr	McKinney *et al.* (1956)
13[2]	8 days	Malancy (1960)
20	1 day	Winter (1962)
0 - 0.6	7 days	Marion (1966)
36	8 days	Malancy (1960)
46	5 day	Marion (1966)
49; 100	7 days; 14 days	Tabak *et al.* (1980)
Extremely slow[c]	-	Chambers *et al.* (1975)

---

[a] All studies used mixed species microbial populations and measured oxygen uptake.

[b] Culture isolated from adapted activated sludge.

[c] Culture isolated from petroleum waste lagoon.

(1977) estimated a refractory index (based on BOD/COD ratios) of 0.23 for benzene, which suggests low susceptibility to treatment. However, this method did not allow for acclimation of the microbial population.

Burns and Roe (1979) measured efficiences of 70-100% (mean = 90%) in field measurements of benzene concentrations at various stages during treatment. Table 4-7 presents these data. Influent concentrations were usually less than 10 μg/l, although levels were as high as 143 μg/l at one plant in Indianapolis. Inhibitory levels for sewage treatment are considerably higher, reported at 1000 mg/l for sludge digestion (U.S. EPA 1977). The fraction of direct loss of benzene attributable to microbial activity cannot be determined from the Burns and Roe (1979) study. However, the combined effect of all wastewater treatment processes appears to be successful in removing benzene from water.

Factors that Limit Biodegradation of Benzene

Certain variables may influence the propensity for or the rate of biodegradation in the environment. These include: benzene concentration, microbial population acclimation, oxygen levels, presence of other nutrients, presence of other hydrocarbons, and the rates of other competing reactions.

- Benzene Concentration. Too high a concentration may repress or inhibit microbial activity; too low a concentration may not induce the required enzymes. Little information is available on these limits, although the lack of microbial activity in a 1%-benzene concentration in 1 ml substrate mixed into 250 ml culture medium was attributed to too high a benzene level. Benzene at 0.1 mg/l inhibited chemotaxis by 50% in a marine *Pseudomonas* (Walsh and Mitchell 1973).

- Acclimation. Numerous studies have reported initial lag periods when populations are first exposed to benzene (Tabak *et al.* 1980, Marr and Stone 1961, Thom and Ag 1975). Enzymes required for degradation are thought to be induced, not constitutive, which supports observations of lag periods (Marr and Stone 1961).

- Oxygen Availability. All reported benzene degradation pathways involve oxidation reactions; anaerobic degradation has not yet been observed. Walker and Colwell (1975) suggested that turbid conditions increase degradation rates of hydrocarbons; in general, a laboratory study on saturated hydrocarbons in crude oil reported a rate of 0.11 mg/day under static conditions compared with 0.24 mg/day in a shake culture. No studies were available on the limitations of oxygen in benzene degradation; however, habitats with low O_2 levels--bottom sediment, deep soil layers, saturated soil, etc.--would not support significant benzene biodegradation by aerobic bacteria.

- Presence of Other Nutrients. The biodegradation of benzene, which only provides a carbon source, may be limited by low concentrations of other nutrients required for microbial growth, such as nitrogen and phosphorus (Howard and Durkin 1974).

- Presence of Other Hydrocarbons. Benzene in the presence of dodecane and/or napthalene in a culture study was degradable, while benzene alone was not (Walker and Colwell 1975). The authors suggested that co-oxidation was necessary for degradation and/or that the benzene concentration was too high in the single hydrocarbon culture. Claus and Walker (1964) suggest that enzymes with similar activities are involved in the metabolism of benzene and toluene. In this laboratory study, toluene-acclimated microorganisms had an advantage, consequently, they were able to immediately degrade benzene while glucose-raised organisms could not.

- Competing Reactions. Other processes controlling benzene concentrations, particularly volatilization, may operate at rates much faster than biodegradation. Faster processes may reduce toxic concentrations to levels supporting metabolism or, in other cases, to levels too low to support metabolism.

4.4.4.3 Soil

Little data are available on chemical or biological fate processes that affect benzene in the soil environment. It is unlikely that any purely chemical transformations of the chemical will occur: benzene is resistant to hydrolysis, to oxidation, and to other chemical reactions except under extreme conditions (e.g., concentrated nitric or sulfuric acid, high temperature).

Versar, Inc. (1975) asserted that benzene in solid waste leachate from landfills "can be degraded during soil migration. Although specific documentation of this degradation has not been found, at least five microbial species, which proved capable of surviving with benzene as their sole carbon source (i.e., they biodegraded the benzene), have been isolated from various soils (see Table 4-21). The biodegradation process has been described in detail in Section 4.4.4.2 and will not be repeated here. Although the other factors cited are important, oxygen is the critical limiting factor for soil degraders. Benzene will volatilize from surface soils faster than organisms can degrade it. Submerged soils, deep subsurface layers of water and soil, cold regions, and other colder seasons in temperate climates are environments where volatilization is slow and, therefore, support persistent benzene levels. Microbial activity, however, would also be reduced under these conditions. No evidence exists for anaerobic degradation of benzene; thus, the compound may persist in low oxygen habitats, such as saturated soil, groundwater aquifers, or lake and estuarine sediments.

TABLE 4-21. MICROBIAL SPECIES ISOLATED FROM SOIL CAPABLE OF DEGRADING BENZENE

Species	Used Benzene as Sole C	Note	Reference
Cladosperium resinae	yes	Growth began >22 days	Cofone *et al.* (1973)
Pseudomonas sp.	yes	Benzene-acclimated organisms showed no lag but organisms raised on glucose did not achieve a maximum rate of oxidation until 190 min.	Claus and Walker (1964)
Achromobacter sp.	yes	"	Claus and Walker (1964)
Pseudomonas putida			Gibson *et al.* (1968)
Nocardia sp.			Wieland *et al.* (1958)
Pseudomonas aeruginosa	yes	Benzene-acclimated organisms showed no lag; glucose-grown organisms exhibited lag	Marr and Stone (1961)
Mycobacterium rhodochrous	yes	"	Marr and Stone (1961)

Rogers et al. (1980) performed laboratory analyses of benzene behavior in montmorillonite clay and two silty clay loams. Absorption and desorption of benzene from three solutions of 10, 100, and 1000 μg/l were measured. It was found that "Montmorillonite clay saturated with Ca sorbed less benzene than the soils; however, Al-saturated clay .... was able to sorb and retain much more benzene than could the soils." These researchers concluded that "sorption of benzene is not the major effect of soil on benzene."

4.4.4.4 Plants

Uptake and metabolism of benzene have been reported for numerous terrestrial plant species (Cross et al. 1979, Howard and Durkin 1974). Degradation pathways more closely resemble those observed in animals than in microorganisms with conversion of benzene to phenol, muconic, fumaric, succinic acids, and phenylalanine. Jansen and Olson (1969) provide the only study that observed the metabolism of benzene for a sufficient length of time. In that study, only a small fraction (0.004 - 0.007%) of benzene was degraded to CO_2; most of it was converted to simple compounds that could be used by plants in metabolic processes. Uptake of benzene from the atmosphere has been observed for water hyacinths, Swiss chard, sugar beet, avocados, potatoes, apples, peppers, and other fruits (Cross et al. 1979, Jansen and Olson 1969); root absorption by tea, laurel, grape and corn plants has also been reported (Howard and Durkin 1974). In the most detailed uptake study, Cross et al. (1979) found an initial lag period before significant degradation occurred, an absorption rate proportional to benzene concentration, and complete transformation of benzene to other compounds (unidentified). Initial atmospheric benzene concentrations were approximately 300 μg/l.

It is difficult to interpolate the results of these experiments to environmental conditions. Certain conditions that are possibly essential to plant degradation of benzene may not occur in the field. For example, atmospheric concentrations high enough and long enough will induce the appropriate plant enzyme systems or support acclimation of an associated microflora.

4.5 SUMMARY

This chapter has described the environmental fate of benzene from the perspective of the three major environmental compartments. The processes that may transfer benzene from one to the other have been analyzed for their significance and reaction rate. The processes that have potential to alter chemically or degrade benzene within a given compartment have been similarly considered.

The major fate processes, both inter- and intramedium, are shown in Figure 4-11 and summarized below.

4.5.1 Intermedium Transfer Processes

4.5.1.1 Air

- Rainout to Water and Land. A process limited first, by benzene's low solubility in water and, second, by the small volume and surface area of precipitation nucleii and droplets.

  Conclusion. Limited overall importance.

4.5.1.2 Water

- Volatilization to Air. Occurs quite quickly; controlled by diffusion within water bodies.

  Conclusion. A major pathway.

- Adsorption to Soils. Occurs on a limited basis; highly dependent on soil type, i.e., organic content, relative concentrations, etc.

  Conclusion. Limited overall importance.

4.5.1.3 Soil

- Volatilization to Air. Occurs quite quickly; controlled by aeration within soil, i.e., enclosed soils will not encourage benzene loss.

  Conclusion. A major pathway.

- Solution into Water. Soil benzene is tightly bound, unbound benzene will dissolve in soil water as determined by its solubility.

  Conclusion. Limited overall importance.

- Runoff to Water. Surficial benzene would be preferentially volatilized. Any benzene bound to surface particles could be carried off physically.

  Conclusion. Limited overall importance.

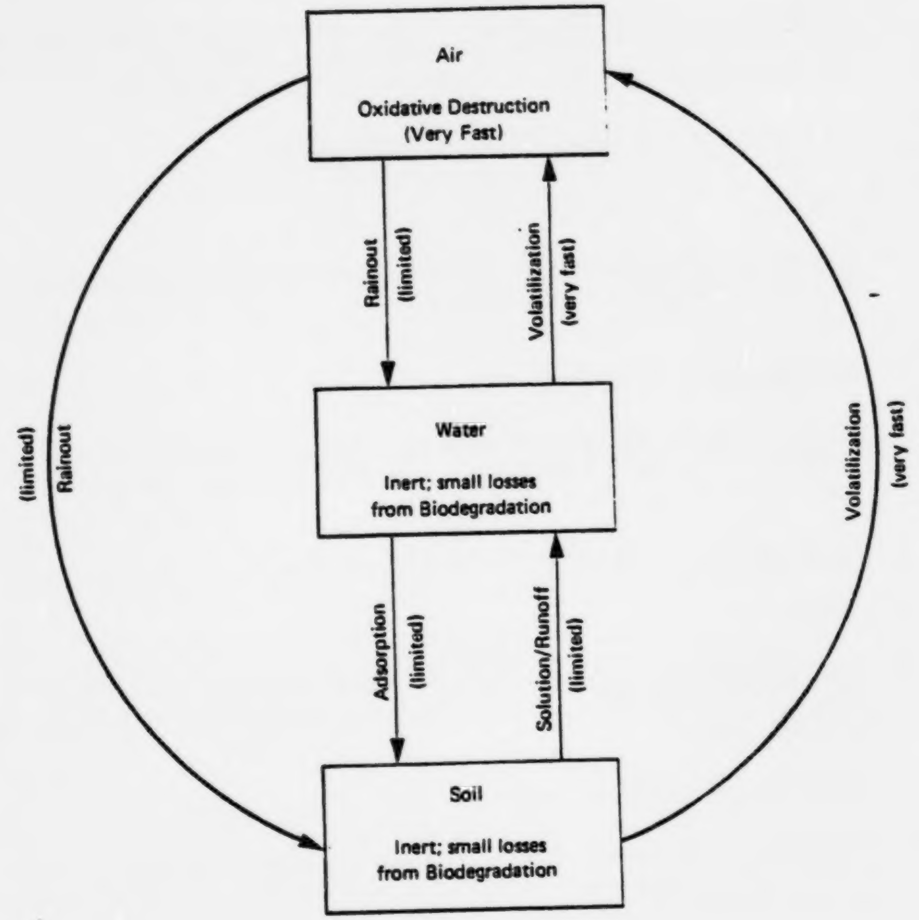

Air
Oxidative Destruction
(Very Fast)
Rainout
(limited)
Volatilization
(very fast)
Water
Inert; small losses
from Biodegradation
(limited)
Rainout
Volatilization
(very fast)
Adsorption
(limited)
Solution/Runoff
(limited)
Soil
Inert; small losses
from Biodegradation

FIGURE 4–11 MAJOR FATE PROCESSES FOR BENZENE

4.5.2 Intramedium Fate Processes

4.5.2.1 Air

- Oxidation by Hydroxyl Radicals. A very fast reaction, determined by concentrations of OH.

  Conclusion. Dominant fate process -- responsible for destruction of most environmental benzene.

4.5.2.2 Water

- Degradation by Microbial Species. Need for initial acclimation periods of up to three weeks and requires presence of appropriate species.

  Conclusion. Important in some habitats, but not universally important.

4.5.2.3 Soil

- Biodegradation. As for water, many factors must be within certain ranges to support this process.

  Conclusion. Important in some habitats, but not universally important.

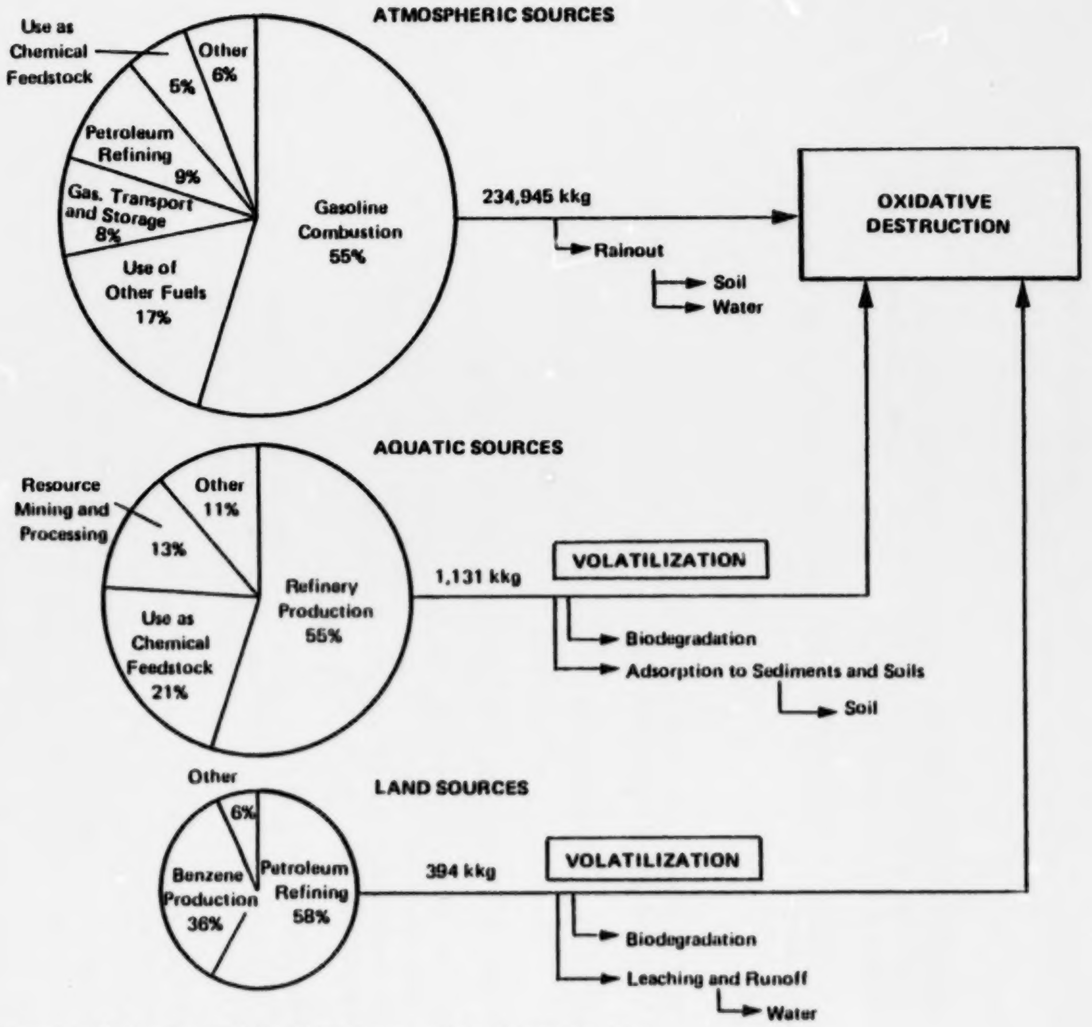
4.5.3 Critical Pathways for Specific Sources of Benzene

The critical pathways for the known releases of benzene to the environment are shown in Figure 4-12. These pathways are called "critical" because they define the processes that act to reduce the total environmental load of benzene.

The three critical pathways are: 1) atmospheric sources → oxidative destruction, 2) aquatic sources → volatilization → oxidative destruction, and 3) land sources → volatilization → oxidative destruction.

Of the total releases of 246,080 kkg in 1978, a maximum of 95.5% may follow the shortest (i.e., no intermedia transfer) pathway--#1, 0.5% may follow #2, and 0.20% may follow #3. Part of these releases will remain in each compartment as ambient or background levels until either biodegradation or entrance to a critical pathway occurs.

It may be concluded that the bulk of benzene releases occurs in the one medium in which they are most speedily broken down and that the small remainder will eventually follow critical pathways to the same ultimate fate; a very small amount of total benzene releases will biodegrade and a similarly small amount will remain dissolved, adsorbed onto sediments, or airborne.


ATMOSPHERIC SOURCES
Use as Chemical Feedstock
5%
Other 6%
Petroleum Refining 9%
Gas. Transport and Storage 8%
Use of Other Fuels 17%
Gasoline Combustion 55%
234,945 kkg
Rainout
Soil
Water
OXIDATIVE DESTRUCTION
AQUATIC SOURCES
Resource Mining and Processing
13%
Other 11%
Use as Chemical Feedstock 21%
Refinery Production 55%
1,131 kkg
VOLATILIZATION
Biodegradation
Adsorption to Sediments and Soils
Soil
LAND SOURCES
Other
6%
Benzene Production 36%
Petroleum Refining 58%
394 kkg
VOLATILIZATION
Biodegradation
Leaching and Runoff
Water

Notes: Processes in boxes and bold type are major fate pathways for any benzene in that particular medium.

Processes that lead to other media are indicated by an arrow, which leads to that medium and implies that all fate processes for that medium apply.

FIGURE 4-12 CRITICAL PATHWAYS FOR BENZENE
(Released Amounts for 1978 Materials Balance)

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5.0 HUMAN EFFECTS AND EXPOSURE

5.1 HUMAN EFFECTS

5.1.1 Pharmacokinetics

For an assessment of chronic toxicity, it is useful to compare and contrast the pharmacokinetics of benzene with that of toluene. These solvents have similar physicochemical characteristics, and where they have been directly compared, they appear to be similar with respect to absorption and distribution in the body. Significant differences do occur in metabolism and this probably accounts for the differences in chronic toxicity.

5.1.1.1 Absorption

Benzene has volatile and highly lipid-soluble chemical characteristics that permit absorption by all exposure routes: dermal, oral and inhalation. The concentration of benzene and the permeability to benzene of the intervening membranes are the principal determinants of the rate of absorption.

Dermal absorption of benzene vapor is presumed to be slow, compared with inhalation at the same air concentration, even though, apparently no one has studied the rate of benzene vapor uptake via the exposed skin surface. Riihimaki and Pfaffli (1978) did study the absorption of toluene across the body surface of human volunteers at a concentration of 2260 mg/m^3. The subjects were exposed for 3.5 hours and their faces were covered with an inhalation mask under slight positive pressure to prevent inhalation uptake. They were clothed only in lightweight pajamas and socks. These researchers calculated total uptake as approximately 26 milligrams, based on a 16% recovery of absorbed dose in the expired air. It is estimated that the same total uptake in 3.5 hours via inhalation would occur at an air concentration of only 38 mg/m^3. (This estimate assumes a respiratory rate of 7 l/min at rest and a respiratory retention factor of 47% for toluene; see the discussion below.) In their experiments on the percutaneous absorption of xylene, Riihimaki and Pfaffli show that uptake is proportional to air concentration. Based on these data and the similarities between xylene, toluene, and benzene, the approximate average skin permeability of the human body to benzene is calculated to be 0.002 $m^3/(m^2 \times hr)$.[2]

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[2] Uptake = 8 mg/hr $\cong$ permeability x 2260 mg/m^3 x 1.8 m^2, where 2260 mg/m^3 was the exposure concentration and 1.8 m^3 is the approximate body surface area.

Dermal absorption of liquid (neat) benzene is much faster than that of vapor, because of the higher concentration and a defatting action on skin that would significantly increase the permeability. Skin absorption of neat benzene was estimated using the data of Sato and Nakajima (1978). In their experiments, human volunteers soaked one hand in neat toluene for 30 minutes. Blood levels after the 1/2-hour exposure reached approximately 25% of the blood levels measured after 1/2-hour inhalation exposure at 376 mg/m^3. It can be shown that blood level is directly proportional to uptake rate, when rate is constant. If it is assumed that the skin absorption rate was constant (an oversimplification, but useful for a demonstration), it is estimated that the uptake rate via the skin of the hand was ∿20 mg/hr.[3] The surface area of the hand is about 2% of the body surface area of 1.8 m^2, or .036 m^2 (Diem and Lentner 1971); therefore, an estimate of absorption rate through skin of liquid benzene is 550 $mg/m^2/hr$.

Absorption of benzene via inhalation is the most important exposure route in the occupational setting, because benzene is highly volatile and absorption into the body via the lungs is rapid. A useful parameter of inhalation absorption is the retention factor, which may be defined as the fraction (or percent) of the inhaled solvent that is absorbed from the inspired air. Most reports indicate that the respiratory retention factor is between 40-50% (U.S. EPA 1978c). Toluene absorption has been more carefully investigated, because of its much lower toxicity, and findings from these studies are extrapolated to benzene exposures. Veulemans and Masschelein (1978a) found that the respiratory uptake rate of toluene was directly proportional to minute volume and concentration. The range of experimental minute volume was varied from a rest rate of 7 l/min to >50 l/min with heavy work, and concentration ranged from 190 to 750 mg/m^3. The retention factor for toluene was 47%. Because of the similar chemical properties of toluene and benzene and the comparable retention factors, the total respiratory uptake of benzene into the body may be estimated by the following equation:

$$\text{uptake (mg)} = \text{concentration } (mg/m^3) \times \text{minute volume } (m^3/min) \times \text{time (min)} \times \text{retention factor } (.5).$$

5.1.1.2 Distribution

In discussing the distribution in the body of a lipid-soluble, water insoluble compound, it is appropriate to view the body as a multicompartmental system. Although each organ may be considered a compartment, it is more usual to treat the body as containing 2-4 compartments, with each compartment made up of organs and tissues having similar pharmacokinetic characteristics. For benzene, a 3-compartment model has generally been adequate to characterize the pharmacokinetics. The first compartment is generally considered to be composed of the blood and highly perfused organs, such as the heart, kidneys, liver, intestines, endocrine glands and brain. This central compartment is the one from which the other compartments, called peripheral compartments, receive drugs and chemicals and from which the chemicals are eliminated from the body. The second compartment is composed of tissues and organs with moderate blood perfusion, such as muscle and skin. The third compartment, especially important in the case of lipid-soluble organics, is composed of slowly perfused tissues, such as fat. Fat differs from most other tissues in having a much higher tissue/blood partition coefficient for organic solvents; i.e., it can accumulate benzene to a greater extent than might be expected on the basis of volume alone. A useful index of the time it takes for the various tissues (or compartments) to reach equilibrium with the central compartment (i.e., the blood, since it can be assumed that rapid mixing occurs within the central compartment) is the saturation half-life, $t_{1/2}$. The saturation half-life depends directly on the volume of the compartment (V_T) and the tissue/blood partition coefficient (λ); and inversely on the blood flow (Q) for the compartment as follows:

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[3] $\frac{376\ mg/m^3}{4} \times .47 \times 0.45\ m^3/hr = 20\ mg/hr$, where $\frac{376\ mg/m^3}{4}$ was the air concentration for equivalent blood levels via inhalation, 0.47 was the respiratory retention factor and 0.45 m^3/hr was the respiratory rate at rest.

$$t_{1/2} = (\lambda \cdot V_T/Q) \times .693.$$

Rough estimates of saturation half-lives for several tissues and the three composite compartments are presented in Table 5-1. Clearly, the distribution to the brain and the central compartment is very rapid. It is so rapid that often it is difficult to delineate this compartment in pharmacokinetic analysis. The third compartment equilibrates so slowly that it usually does not reach saturation equilibrium with the blood during continuous exposure, such as an 8-hour occupational exposure. For the same reason, a tendency for "baseline" blood levels to build up over continuous day-to-day exposure could occur as a result of this third compartment being similarly slow.

Blood levels usually cannot be used to quantitate absorption unless exposure conditions in terms of both concentration and time are known. During inhalation exposure, blood levels rise rapidly to a "quasi"-steady state, reflecting rapid absorption and slow metabolism and distribution to other tissues. When exposure is terminated, blood levels fall rapidly at first, reflecting continued distribution to the rest of the body as well as metabolism and elimination. After an initial rapid decline, slower phases of decline are noted, because elimination is rate limited by the transfer of the chemical from the peripheral compartments into the central compartment.

Sato et al. (1974) studied and compared the pharmacokinetics of benzene and toluene in human volunteers. The decline in blood levels

TABLE 5-1. ESTIMATES OF THE SATURATION HALF-LIFE OF TOLUENE AND BENZENE BETWEEN BLOOD AND TISSUE

	λ[a]		V_T/Q[b]	$t_{1/2}$[c]	
	Benzene	Toluene		Benzene	Toluene
Compartment 1	1.5	2	1.5	1.5	2
Liver	1.6	2.6	2.5	2.8	4.5
Kidney	1.1	1.5	.24	0.2	0.2
Brain	1.9	3.0	1.3	1.7	2.7
Compartment 2	1.1	1.2	17 (resting)	13	13
Compartment 3	50	100	47	1630	3200
Fat	58	113	50	2000	3900
Marrow	16	35	25	390	850

[a] λ = Tissue/blood partition coefficients obtained from data of Sato _et al._ (1974).

[b] V_T/Q = Volume of tissue/blood flow (ml/ml/min) from Papper and Kitz (1963).

[c] $ts/_2$ = Saturation half-life = $.693 \times V_T/Q \times \lambda$ (minutes).

after a 2-hour exposure to either benzene (at 80 mg/m^3) or toluene (at 377 mg/m^3) was followed for 5 hours. The equations that describe the decline in blood levels are sums of three exponentials as follows:

benzene,

$$y = 0.0593e^{-0.418t} + 0.086e^{-0.0238t} + 0.0287e^{-0.00347t};$$

toluene,

$$y = 0.355e^{-0.355t} + 0.352e^{-0.0197t} + 0.129e^{-0.00339t};$$

where t is the time in minutes and y is blood concentration in mg/l.

These model equations together with other data indicate that benzene and toluene are absorbed and distributed into the body quite similarly. The exponents of the equations are similar to a striking degree. Also, the coefficients of the toluene equation are about 4-6 times higher than the respective coefficients in the benzene equation, which follows from the fact that the toluene exposure concentration was 4.7 times the benzene exposure concentration.

In an important respect, the equations are probably misleading for both toluene and benzene, because they suggest no appreciable accumulation of either solvent from day to day. Data reported by Konietzko et al. (1980) and theoretical considerations indicate that accumulation can occur on a day-to-day basis. Konietzko monitored exposure concentrations and blood concentration levels at the beginning and end of each 8-hour work day over a 2-week period in workers occupationally exposed to toluene. These data are reported in Table 5-2. An apparent upward trend in the toluene blood concentration values occurs each morning before exposure over the 5-day work week. The lowest levels were measured on Monday mornings. The half-life of the terminal phase of elimination would have to be on the order of 2000 minutes (30 hours) for baseline blood levels to build up as they appeared to do in the exposed workers. This half-life is comparable with the theoretical saturation half-life for fat given in Table 5-1. The terminal phase half-life calculated from the equations of Sato et al. (1974) are on the order of 200 minutes. This finding of Sato and coworkers is understandable because the exposure was only for 2 hours in their experiments and the blood concentration data were only determined for 5 hours after the exposure. These time periods are too brief to delineate a very slow elimination phase.

In summary, benzene is absorbed into the body regardless of the route; the major difference among routes is the rate of absorption. Once benzene is into the blood, it is distributed widely to all tissues. The relative rate of uptake into each tissue is determined by the relative perfusion of the tissue by blood. Accumulation in fat is slow because of low perfusion; however, the potential uptake is high because of the lipid solubility of benzene.

TABLE 5-2. TOLUENE CONCENTRATIONS IN AIR AND BLOOD

		Monday	Tuesday	Wednesday	Thursday	Friday
First Week	Toluene in air (ppm)	225 (95-303)	233 (153-383)	209 (107-341)	212 (92-314)	203 (124-309)
	Toluene in blood before exposure (μg/ml)	0.12 (0.09-0.24)	- - -	0.51 (0.28-0.82)	- - -	0.77 (0.29-1.67)
	after exposure	3.63 (2.3-4.75)	- - -	6.69 (4.21-10.36)	- - -	6.70 (3.39-10.67)
Second Week	Toluene in air (ppm)	285 (145-473)	304 (190-521)	309 (213-413)	232 (125-451)	191 (105-432)
	Toluene in blood before exposure (μg/ml)	0.27 (0.07-0.57)	- - -	1.00 (0.35-1.51)	- - -	1.21 (0.44-2.29)
	after exposure	11.60 (6.99-17.10)	- - -	10.29 (3.24-20.31)	- - -	5.85 (1.94-9.78)

Range in parenthesis and means of data for eight persons.

Source: Konietzko _et_ _al._ (1980).

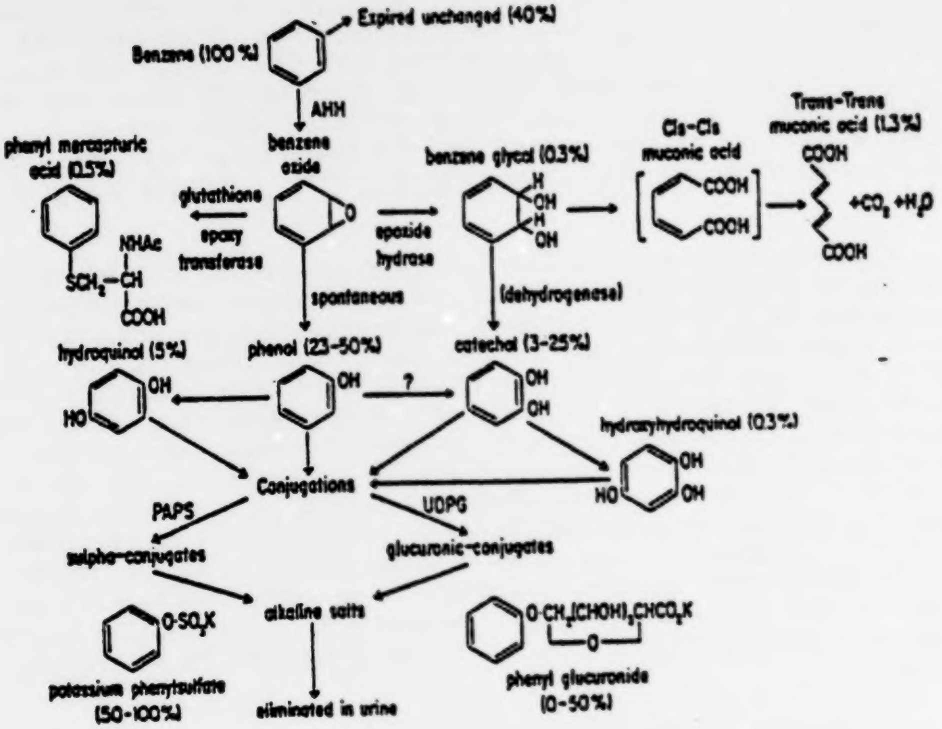
5.1.1.3 Metabolism and Excretion

The metabolism of benzene has been studied in several mammalian species. Figure 5-1 indicates the pathways that have been identified. A large fraction of absorbed benzene is excreted unchanged in the expired air. The actual proportion excreted unchanged varies among species and also depends on the dose. Andrews and coworkers (1977) reported that 70-85% of a subcutaneously administered dose (880 mg/kg) was expired in mice. Parke and Williams (1953a, b) found 40-50% of an oral dose (150-500 mg/kg) in the expired air of rabbits. Estimates of the fraction excreted in the expired air of humans range between 12 and 50% (Teisinger _et al._ 1952, Srbova _et al._ 1950, Nomiyama and Nomiyama 1974).

Alternatively, a mixed function oxidase system, which is associated with the microsomal fraction of tissue homogenates, oxidizes benzene. This system is a group of enzymes; the specificity and activity of which varies considerably with the species and tissue. Gonasun and coworkers (1973) described this mixed-function oxidase system from mouse, rat, and rabbit liver microsomes. It required a NADPH generating system and oxygen for activity and contained cytochrome P-450. Pretreatment of the animals with benzene increased the _in vitro_ activity of the system about 80%. The mouse derived system was about 10 times as active for oxidizing benzene on a per milligram of protein basis as that from rat and rabbit. Generally, the liver contains a highly active mixed-function oxidase system that is easy to prepare in useable quantities; however, other tissues also contain active systems that contribute to benzene metabolism and may figure prominently in understanding mechanisms of toxicity.

The product of the initial reaction of benzene with the mixed-function oxidase is the intermediate, benzene epoxide -- an unstable, reactive metabolite, which may undergo a variety of interactions with cellular constituents. Only three pathways for which there is evidence are shown in Figure 5-1. The major route is to phenol, which occurs by a nonenzymatic reaction (Jerina _et al._ 1968, Snyder and Kocsis 1975). The fraction of benzene that is metabolized to phenol also shows species and dose dependent variation. Parke and Williams (1953a, b) found that about 20% of an oral dose (150-500 mg/kg) was excreted in the urine of rabbits as conjugates of phenol. The much lower ratio of catechol/phenol metabolites when rabbits were dosed with phenol compared with the ratio when rabbits were dosed with benzene (i.e., ∿.01 and .08, respectively) suggests that catechol was derived primarily from phenol.

A second route of degradation of the epoxide is catalyzed by the enzyme epoxide hydrase, which oxidizes the intermediate to trans-1,2-dihydro-1,2-dihydroxybenzene (benzene glycol). Jerina and coworkers (1968) identified this enzyme in both microsomal and soluble fractions of rabbit liver homogenates. The soluble fraction of liver homogenates dehydrogenated the 1,2 glycol to catechol. Previously, Parke and


Expired unchanged (40%)
Benzene (100 %)
AHH
benzene oxide
phenyl mercapturic acid (0.5%)
glutathione
epoxy transferase
NHAc
SCH2–CH
COOH
epoxide hydrase
benzene glycol (0.3%)
H
OH
H
OH
Cis-Cis muconic acid
COOH
COOH
Trans-Trans muconic acid (1.3%)
COOH
COOH
+CO2 +H2O
spontaneous
(dehydrogenase)
hydroquinol (5%)
phenol (23–50%)
catechol (3–25%)
OH
HO
OH
?
OH
OH
hydroxyhydroquinol (0.3%)
OH
HO
OH
Conjugations
PAPS
UDPG
sulpho-conjugates
glucuronic-conjugates
alkaline salts
O-SO3K
potassium phenylsulfate (50-100%)
eliminated in urine
O-CH2(CHOH)3CHCO2K
O
phenyl glucuronide (0-50%)

[a]Percentages are approximate values and do not necessarily apply to humans.

NOTES:
AHH = aryl hydrocarbon hydroxylase
UDPG = uridine diphosphate glucuronyl transferase
PAPS = 3′-phospho-adenosin-5′-phosphosulfate

Source: U.S. EPA (1978b)

FIGURE 5–1 METABOLIC PATHWAY OF BENZENE IN LIVER[a]

Williams (1953a, b) had shown that the muconic acids were only found in rabbits dosed with benzene but not in those dosed with phenol.

The third route of metabolism via the benzene epoxide is conjugation of the intermediate with glutathione, presumably via the glutathione-S-transferase system. A phenyl mercapturic acid is the eventual product after the elimination of glycine (Jerina et al. 1968, Goldstein 1974).

In contrast to benzene metabolism, toluene is preferentialy oxidized at the methyl group of benzyl alcohol. Approximately 80% is metabolized via benzyl alcohol, while another 16% is excreted unchanged in expired air (Veulemans and Masschelein 1978a, b, 1979). A small percentage of toluene is metabolized via a reactive intermediate, i.e., toluene epoxide to cresols and methylphenyl mercapturic acid (Dean 1978, Van Doorn et al. 1980).

Several compounds have been shown to inhibit benzene metabolism, presumably by competitive inhibition of the mixed function oxidase system. Benzene metabolism was inhibited by compounds known to interact with the mixed function oxidase system, such as aniline, metyrapone, aminopyrine, SKF-525A, and cytochrome c (Gonasun et al. 1973). Toluene inhibited benzene metabolism in vitro of the 10,000 G supernatant fraction, which contains the microsomal enzymes (Sato and Nakajima 1979). These researchers demonstrated in rats a dose-dependency of the fraction of benzene excreted as total phenol, from 41% of dose at 0.3 mmol/kg (24 mg/kg) to 8% of dose at 5 mmol/kg (390 mg/kg). Coadministration of toluene at 5 mmol/kg reduced the percent of benzene (at 5 mmol/kg) excreted as phenol to about 1% of the dose. Their work indicates a reciprocal competitive inhibition between benzene and toluene in which toluene is a far more effective inhibitor of benzene than benzene of toluene. Inhibition was most apparent at high dose levels. Human volunteers showed no inhibition of benzene by toluene or toluene by benzene following coexposure to 376 mg/m^3 toluene and 80 mg/m^3 benzene.

Results of the studies of Andrews et al. (1977) and Irons et al. (1980) show that metabolism of benzene occurs in other tissues as well as in the liver, especially in the bone marrow. Using various extraction procedures to differentiate benzene from benzene metabolites in bone marrow, Andrews showed that metabolites were nine times higher in marrow than in blood and six times higher than in the liver after benzene was injected (880 mg/kg s.c.) in mice. They showed that the bone marrow was unable to concentrate phenol, phenyl glucuronide or phenyl sulfate to this extent when these metabolites of benzene were injected. The simultaneous injection of toluene with benzene markedly reduced the concentration of benzene metabolites in the tissues, including marrow, fat, spleen, liver, and blood. However, tissue levels of nonmetabolized benzene were not as markedly altered by simultaneous injection of toluene.

5.1.2 Acute Effects

Benzene is a general central nervous depressant in acute doses. The nature and extent of specific acute symptoms have shown marked variations between individuals. This probably reflects, for the most part, differences in the rate of uptake (exposure concentration) and the extent of uptake (exposure duration).

Death has resulted from single exposures to benzene in air at concentrations of 20,000 ppm (64,000 mg/m^3) within 5 to 10 minutes. Symptoms proceeded through headache, nausea, staggering gait, paralysis, convulsions, unconsciousness and death. Death may result from respiratory arrest or cardiovascular collapse. Severe but nonfatal acute exposures have produced similar symptoms. Less severe exposures may produce giddiness and euphoria (U.S. EPA 1980).

Death may be due to cardiac arrhythmias resulting from sensitization by benzene of the heart muscle to catecholamines . Thus, the danger of fatality from benzene exposure may be increased during periods of physical activity and stress (Snyder and Kocsis 1975).

5.1.3 Chronic Effects

The most important effect resulting from chronic benzene exposure is hematotoxicity. Extensive discussions of the literature on hematotoxicity of benzene have been presented in recent reviews (U.S. EPA 1978a, b, c; 1980, Snyder and Kocsis 1975, and others listed in U.S. EPA 1980). This discussion is limited to a brief review of the hematological disorders considered to represent the most significant hazards associated with chronic benzene exposure and for which risk estimates might be calculated. These disorders are pancytopenia (and closely related phenomena) and leukemia.

5.1.3.1 Pancytopenia

Pancytopenia is a deficiency of all cellular elements of the blood. In benzene poisoning, the deficiency results from an inadequate production of the several blood cell types, i.e., cytopenia associated with hypoplasia of the bone marrow or occasionally with a hypercellular marrow exhibiting ineffective hematopoiesis. In its less severe forms, specific deficiencies may occur in blood elements (e.g., anemia, leucopenias, or thrombocytopenia). Deficiencies in each element lead to certain symptoms, such as hemorrhagic conditions resulting from thrombocytopenia or susceptibility to infection because of leucopenias. In its severe form,a pancytopenia caused by benzene poisoning is usually associated with aplastic anemia.

Systematic studies of occupationally-exposed workers where determinations of benzene levels were also performed are summarized in Table 5-3. The causal relationship between benzene and pancytopenia in humans is strongly supported by several studies (Greenburg _et al_. 1939,

TABLE 5-3. SUMMARY OF BENZENE EXPOSURE AND RELATED HEMATOTOXICITY

Industry	Number of Employees	Incidence of Hematological Toxicity	Level of Exposure (mg/m^3)	Duration of Exposure	Reference
Rotogravure Printing	332 exposed 82 controls	65[a]	35-3380 420 median	3-5 yr	Greenburg et al. 1939 Goldwater 1941 Goldwater and Tewksbury 1941
Rubber Factory	1104	108[b]	1595 peak 319 ave.	NR	Wilson 1942
Raincoat Factory	184	60[c]	437-695	NR	Helmer 1944
Rubber Coating Plant	32	5[d]	80-319	NR	Pagnotto et al. 1961
Shoe Manufacturing	217 100 controls	51[e]	96-670	3 mos-17 yr	Aksoy et al. 1971
Shoe Manufacturing	NR	32[f]	478-1914	4 mos-15 yr	Aksoy et al. 1972
Chemical Factory	365	~40%[g]	32-130	< 1 yr	Doskin 1971

[a] Twenty-three of the 65 showed severe signs of hematotoxicity, requiring hospitalization. Compared with control, the most frequently observed findings were anemia, macrocytosis and thrombocytopenia. After benzene was replaced with other solvents, hematological recovery was demonstrated.

[b] Eighty-three had mild hematological effects; 25 had more severe pancytopenia -- 9 of the 25 were hospitalized and of the 9, 3 died.

[c] Thrombocytopenia and cutaneous hemorrhages were reported. Reevaluation 16 months after cessation of benzene use revealed the recovery of 46, 12 still had significant effects, and 2 died.

[d] Hemoglobin levels reduced. One of the 5 had high phenol excretion, said to correspond to 185 mg/m^3 benzene exposure.

[e] Leucopenia and/or thrombocytopenia was found in 41 workers, basophilia, eosinophilia or lymphocytosis was found in the 10 others.

[f] Thirty-two workers suffered from pancytopenia associated with benzene exposure. Bone marrow studies revealed cases of hypoplastia, hyperplasia, and instances of large erythroid precursors.

[g] Mild thrombocytopenia was the most common abnormality. Mild anemia, lymphocytosis, a biphasic leucocyte response or bone marrow hypercellularity were reported for some exposed workers. Benzene levels and monitoring procedures were not clearly defined.

NR - not reported.

Goldwater 1941, Goldwater and Tewksbury 1941, Helmer 1944), which showed that the hematological effects essentially ended when benzene was replaced with another solvent.

These studies strongly implicated benzene as a major cause of hematological disorders. A more definite interpretation, however, especially with regard to a dose-effect relationship is difficult. One reason is that in the occupational setting, workers have had widespread exposure to other solvents. Though these solvents may not be hematotoxic themselves, they could interact with benzene in the body and perhaps alter its metabolism and thereby affect its toxicity.

The most important issue is that it cannot necessarily be assumed that the average concentrations of benzene measured in a workplace actually indicate the average exposure dose to each worker. It is much more likely that there is a wide variation in absorbed doses among workers because of variations in work habits and tasks. Furthermore, it is likely that most incidences of hematological disorders are associated with the higher exposures to benzene. The essential data for estimating a dose-effect relationship are missing, unless the individual's exposure dose can be compared with the occurrence of disease in that individual. Thus, while the epidemiological data tend to indicate that benzene can cause hematological disorders, they cannot be applied to describe the dose-effect relationship.

The hematotoxicity of benzene has been extensively studied in experimental animals. Frequently, it has been reported that benzene causes leucopenia (i.e., decreased white blood cell counts) in experimental animals. The U.S. EPA notes problems in the interpretation of studies reporting depression of white blood cell counts (U.S. EPA 1978b). White blood cell counts vary considerably among species, with stress, age, and among individual animals of the same species. The U.S. EPA's report points out that many studies have inappropriate controls. Furthermore, it is unclear whether depressed white blood cell counts truly reflect bone-marrow damage. Though leucopenia may be difficult to specify and, in general, is a nonspecific index of disease, in the case of benzene, leucopenia has generally been shown to be an early indicator of toxicity reflecting depression of hematopoietic tissue. A dose-response relationship has been demonstrated (Wolf et al. 1956, Deichmann et al. 1963). Table 5-4 summarizes results from a number of studies on the leucopenic effect of benzene. For comparative purposes, the exposure doses have been recalculated in some cases on a mg/kg/day, 5 day/week basis. To recalculate from inhalation exposure, appropriate minute volumes for the species, body weights, and an assumed retention factor of 50% were used as shown in the two examples. Based on these data, it is estimated that the threshold for the leucopenic effect in laboratory animals is between 1 and 10 mg/kg/day, administered over an extended period of time. Following a nonlethal exposure to benzene in experimental animals, hematological disorders generally return to normal.

TABLE 5-4. LEUCOPENIC EFFECTS OF BENZENE

Species	Route/Duration	Lowest Effect Level	No Effect Level	Reference
Rat	Inhalation/8 hr/d, 5 d/wk, 20 wk	158 mg/m^3 (~12.6 mg/kg/day)[a]	- - -	Nau *et al*. 1966
Rat	Gavage/132 days	10 mg/kg/day	1/mg/kg/day	Wolf *et al*. 1956
Rat	Subcutaneous/14 days	440 mg/kg/day	- -	Matsushita 1966
Dog	Inhalation/5-8 hr/d, 4-5 d/wk ~ 12 weeks[c]	2500 mg/m^3 (~ 240 mg/kg/day)[b]	- -	Hough *et al*. 1944
Rabbit	Subcutaneous/2 wk	0.2 ml/kg/day	- -	Kissling and Speck 1972
Rat	Inhalation/5-6 hr/d, 4 d/wk 31 wk	145 mg/m^3 (~ 8 mg/kg/day)	95 mg/m^3 (~ 5 mg/kg/day)	Deichmann *et al*. 1963
Rat Guinea Pig Dog	Inhalation/continuous for 127 days		56 mg/m^3 (~ 16 mg/kg/day)	Jenkins *et al*. 1970

[a] $.5 \times .0001\ \mathrm{m^3/min} \times 60\ \mathrm{min/hr} \times 8\ \mathrm{hr} \times 158\ \mathrm{mg/m^3} \times \frac{1}{.3\ \mathrm{kg}} \cong 12.6\ \mathrm{mg/kg/day}.$

[b] $.5 \times .005\ \mathrm{m^3/min} \times 60\ \mathrm{min/hr} \times 6.5\ \mathrm{hr} \times 2500\ \mathrm{mg/m^3} \times \frac{1}{10\ \mathrm{kg}} \cong 240\ \mathrm{mg/kg/day}.$

[c] Leucopenia was evident by this time.

Other results, summarized in greater detail in the U.S. EPA (1978b), are consistent with the evidence that benzene exposure is hematotoxic; benzene depresses iron incorporation into erythrocytes, granulocyte precursor activity, and DNA synthesis in bone marrow. Studies by Lee _et_ _al_. (1974), Uyeki _et_ _al_. (1977), Moeschlin and Speck (1967), and others have deomonstrated that the toxic effect is on proliferating cells, i.e., cells undergoing division and differentiation. Nonproliferative cells (e.g., reticulocytes) or nonproliferating cells (e.g., resting stem cells) appear to be relatively resistant to benzene toxicity.

5.1.3.2 Leukemia

Leukemia can be defined as a neoplastic proliferation and accumulation of white blood cells in blood and/or bone marrow. The four main types of leukemia include: acute and chronic myelogenous (also known as granulocytic) leukemia, and acute and chronic lymphocytic leukemia. In addition, other types of leukemia are related to these four major types. There is some disagreement concerning the diagnostic criteria. Erythroleukemia, acute promyelocytic leukemia, stem cell leukemia and acute myelomonocytic leukemia, all of which have been associated with benzene exposure, are generally considered to be variants of acute myelogenous leukemia (U.S. EPA 1978b).

The concept that benzene could induce leukemia in humans required considerably more time to develop than the idea that benzene produced aplastic anemia or pancytopenia. This resulted because of the early success in demonstrating benzene-induced bone marrow depression, but a failure to produce leukemia in experimental animals with benzene. Moreover, there were diagnostic problems in identifying leukemias in populations of workers subject to bone marrow depression.

It was estimated that the literature contained references to approximately 150 cases of benzene-associated neoplastic disease; and the best documented of these occurred in industries where chronic benzene poisoning has been detected. It was observed that, "Very often leukemia develops in subjects with benzene-induced hyporegenerative anemia or pancytopenia of more or less longstanding duration and constitutes the acute terminal stage of the disease. The leukemia might become clinically apparent only a few weeks before death; in these cases, the anemia can be considered as being a pre-leukemia stage." (Vigliani and Forni 1976).

It is generally recognized that severe bone marrow depression may predispose to leukemia. Vigliani and Forni (1976) cited 83 cases of benzene hemopathy observed in Italy; 14 of these deaths were due to aplastic anemia and 18 were due to leukemia. In a recent review of 44 pancytopenia patients, Aksoy and Erdem (1978) noted that six had developed leukemia. It is significant that these cases of leukemia occurred in industries where workers were exposed to atmospheric benzene concentrations high enough (600-1200 mg/m^3) to produce bone marrow depression (U.S. EPA 1978b, 1980).

Recent studies by Askoy and coworkers in Turkey are supportive of the causal relationship between benzene exposure and leukemia (Aksoy et al. 1974a, b, c, 1976a, b, Aksoy 1977). Individual case study reports are given for workers with aplastic anemia that progressed through a preleukemic stage and culminated in acute leukemia or erythroleukemia. Aksoy observed 26 patients with acute leukemia. These employees were using benzene solvent during shoe manufacturing operations with occupational exposures in the range of 670-2073 mg/m^3 with durations ranging from 1-15 years. Among the 26 patients with leukemia, 14 cases were acute myeloblastic leukemia, 4 preleukemia, 3 acute erytheroleukemia, 3 acute lymphoblastic leukemia and 1 each was acute promyelocytic and acute monocytic leukemia.

The 26 cases of leukemia or preleukemia were observed in a group of 28,500 workers over an 80-month period from 1966-73. These cases were calculated to give an annual incidence rate of 13 per 100,000, which is significantly greater ($p < 0.02$) than the rate of 6 per 100,000 for the general population. The control incidence was apparently derived from the incidence in developed nations, rather than being specific to Istanbul, which is an uncertain comparison. Counterbalancing this cause of uncertainty is the fact that the age and type distribution of leukemia strongly differ from that in the general population; the incidence of acute myeloblastic leukemia and its variants was much greater than in the general population and the average age of diagnosis (34.2 years) younger than for the general population. Estimates of exposure levels are highly uncertain; however, the U.S. EPA (1978a) report estimated an average exposure level of 63.6 ppm ($\sim$200 mg/m^3) over a 10-hour day, 6-day week, 9.7-year average duration of employment.

In a series of epidemiological studies, the Occupational Health Studies Group of the University of North Carolina investigated the causes of mortality in a 10-year experience of male workers at four tire manufacturing plants. Their findings are summarized in the U.S. EPA (1978b). Out of 5106 deaths, 1014 were due to all cancers. Deaths due to cancer of the lymphatic and hematopoietic system were 31% higher than expected and were increased in cohorts of each of the four companies. The standard mortality ratios (SMR) for several categories of cancer associated with the hematopoietic system were: 129 for the category lymphosarcoma and Hodgkin's disease; 130 for all forms of leukemia; 158 for lymphatic leukemia; and 291 for lymphatic leukemia in the age group 40-64. These studies did not evaluate coexposure to other leukemogens and other environmental toxic agents. Little or no data were available to estimate the levels of exposure to benzene.

Infante and coworkers (1977a, b) collected data on 748 workers employed in Pliofilm ® production at plants in St. Mary's and Akron, Ohio, from January 1, 1940 to December 31, 1949. The authors succeeded in obtaining data on the vital status of 75% of the employees from January 1, 1950 through June 30, 1975. The work is concerned with death due to malignancy of the hematopoietic and lymphatic system and leukemia and clearly demonstrates a significant increase in the SMR for these diseases among the workers studied compared with two control populations. Control 1 was U.S. white male general population standardized for age and time period over which the study cohort lived. Control 2 consisted of 1447 white men who had been employees in Ohio at a fibrous-glass construction products factory between January 1, 1940 and December 31, 1949. Using updated data reported in the Carcinogen Assessment Group's (CAG) report (U.S. EPA 1978a), 9 deaths resulted from all forms of leukemia in the two occupationally-exposed groups, where the expected incidence was 1.25, for a standardized mortality ratio of 720. The SMR was greater when cases of chronic myelogenous leukemia were excluded from consideration. Some do not consider chronic myelogenous leukemia to be linked with benzene (U.S. EPA 1978b).

Estimates of the exposure levels to benzene of the study group are highly debatable and must be regarded as uncertain. Infante *et al.* (1977a, b) argue that benzene was the only solvent that could be responsible for the increases in leukemia and that benzene levels averaged between 32-50 mg/m^3, based on a 1946 survey. However, other sources suggest levels may have been considerably higher.

According to testimony before the Occupational Safety and Health Administration (OSHA), levels exceeded 700 mg/m^3 in certain plant areas (Harris 1977). Several references cited by Tabershaw and Lamm (1977) in a letter to the editor in response to the study by Infante *et al.* (1977a) also indicate that the exposure levels in these plants were probably greater than the prevailing standards of the times during the 1940s; i.e., they may have been in the 300-3000 mg/m^3 range.

The Carcinogen Assessment Group's final report (U.S. EPA 1978a) assumed that the average worker exposure was the same as the prevailing recommended maximum limits for the years 1940-75. They calculated a time-weighted average occupational exposure for the 36-year period of 23.3-39.9 ppm (74-127 mg/m^3). Although this estimate is higher than the estimate of Infante and coworkers, it still may not be high enough for two reasons. First, benzene levels were not monitored in one factory from 1940-46. In 1946, new ventilation equipment was installed after which a survey showed "most areas" in the plant ranged from 0-15 ppm (0-48 mg/m^3) (U.S. EPA 1978a). Prior to 1946, the CAG estimate of benzene levels is only 15-100 (48-319 mg/m^3), which Tabershaw and Lamm (1977) have already pointed out as appearing unrealistically low. Second, the estimate of the CAG group includes the years between 1957-75, when maximum permissible limits had been significantly reduced, but when most workers in this study probably had already left the plants because of retirement or new jobs (see discussion below).

Hattis and coworkers (1980) suggested that the duration of exposure used in the CAG report of 25-36 years overstates the exposure; it is longer than the average length of exposure in the study by Ott et al. (1978) of approximately 9 years, and longer than the average employment tenure in the plastics and rubber industries of 7.1 years (BLS 1966). The likely overestimate for the duration of exposure may tend to compensate for the possible underestimate in concentration levels; however, the uncertainty in the overall exposure estimates should not be underemphasized. Moreover, as noted in connection with the association between pancytopenia and benzene exposure, it is likely that the average exposure concentration does not represent, but actually underestimates, the actual exposure of those workers who later developed disease. The CAG report (U.S. EPA 1978a) considers this study the best available data upon which to base risk estimates.

The CAG (U.S. EPA 1978a) also used another recent benzene-related mortality study by Ott et al. (1978) to make a risk estimate. Out of 594 workers exposed to benzene, 3 died from nonlymphocytic, nonmonocytic leukemia, where 0.8 was expected (R=3.75). Because of the low number of deaths evaluated, the increase is of borderline statistical significance. The time-weighted average benzene exposure for the three deaths resulting from leukemia was characterized as low (6-29 mg/m^3) or very low (< 6 mg/m^3). The CAG analysis estimated that the average exposure in terms of mg/m^3 times months = 1941 and an average lifetime estimate of .55 mg/m^3. As with the Infante studies, the large degree of uncertainty in actual benzene exposures to workers who later developed leukemia should not be underrated.

The foregoing data are suggestive of a relationship between benzene exposure in industries where benzene has been used as a solvent and the occurrence of leukemia in those industries. Epidemiological data from industries where benzene is either produced or is used as a reactant in chemical syntheses, such as the petroleum-petrochemical industry or in coke-oven operations, have yielded data which, by contrast, do not indicate an increased incidence of leukemia.

Thorpe (1974) reported on a study of 38,000 workers in eight Esso-affiliate plants during the 10-year period, 1962-71. Forms were sent to each of the affiliates requesting data on a variety of factors related to several problems; the most important of which related to the incidence of aplastic anemia and leukemia. The incidence of aplastic anemia was too low to permit accurate statistical comparisons between the workers and the control group. The incidence of leukemia in the general population was reported to be 3-8/100,000. Overall, 18 cases of "leukemia" were reported in the test group; however, exact diagnoses of the types of leukemia were not available. It could not be demonstrated that the incidence of leukemia in these plants exceeded the incidence in the population at large.

Benzene is also produced in the steel industry as a by-product of the coking process. The health status of coke-oven workers has been of continuing interest and Redmond et al. (1976) pursued a longitudinal study of the mortality among 58,828 workers in steel plants in western Pennsylvania. Data include records of 8628 deaths and the results showed that coke plant workers exhibited a greater risk of respiratory cancer and kidney cancer than the general population of steel workers. Any indication of cancer of the lymph or hematopoietic organs is significantly lacking from these data.

Animal experimental results weakly support the view that benzene is leukemogenic. The best available data in terms of experimental design, adequacy of reporting, and duration of the study are summarized in Table 5-5. Statistically significant results were obtained by Snyder et al. (1980) in C57BL/6J mice -- a strain that carries a virus, which makes these animals much more susceptible to induction of lymphoma following exposure to radiation, carcinogens, or immuno-suppressive agents. Of the eight animals that died with hematopoietic neoplasms, six were from lymphocytic lymphoma in which there was thymic involvement, one with plasmacytoma (myeloma) and one with leukemia (predominant cell type appeared to be hematocytoblast). The two control animals died of lymphocytic lymphoma without thymic involvement. In contrast, results with AkR strain mice were negative with respect to increased incidence of hematopoietic neoplasms, although this strain is also susceptible to lymphoma. A lower exposure level was necessary because of very poor survival of this strain at the 950 mg/m^3 exposure level.

Maltoni and Scarnato (1979) reported a statistically significant increase in zymbal gland carcinomas. Zymbal gland carcinomas are reported to be rare in untreated rats; however, they are readily induced by systemically administered carcinogenic agents (Baker et al. 1979). A nonstatistically significant, increased incidence of leukemias, apparently more pronounced in male rats, was also reported.

5.1.4 Summary of Effects on Humans

5.1.4.1 Ambient Water Quality Criteria -- Human Health

Because benzene is suspected as being a human carcinogen and no recognized safe concentration exists for a human carcinogen, the recommended concentration of benzene in water is zero (U.S. EPA 1980).

This water quality criterion is based on the human epidemiological data (Askoy 1977, Infante et al. 1977a, b, Ott et al. 1978), and is supported by the animal experimental data in Sprague-Dawley rats (Maltoni and Scarnato 1979). These epidemiological studies were used by the U.S. EPA to recommend a target water level of 8 μg/l to keep any additional lifetime cancer risk below 10^{-5}. The 8 μg/l level is based on an equivalence in response to an absorbed dose of 16.2 μg/day for a human lifetime, regardless of the route. This target-water level was predicted to give an incremental lifetime risk of leukemia of 10^{-5}. Further details of the derivation are given in the Appendices to the criteria document for benzene (U.S. EPA 1980) and the CAG report (U.S. EPA 1978a), and is also discussed in Section 5.1.6 below.

TABLE 5-5. CARCINOGENICITY OF BENZENE IN EXPERIMENTAL ANIMALS

		Results				
Species	**Route/dosage**	**Neoplasm Type**	**Test**		**Control**	**Reference**
Mice, C57BL/6J[a] 65 ♂	Inhalation/950 mg/m^3 6 hr/d, 5 d/wk for lifetime	Hematopoietic neoplasms	8/40[b]		2/40	Snyder *et al.* 1980
		Bone marrow hyperplasia without evidence of hematopoietic neoplasm	13/32[b]		0/38	
	(∿200 mg/kg/day for lifetime)	Spleen hyperplasia without hematopoietic neoplasm	16/32[b]		2/38	
AkR[a] 5 ♂	Inhalation/300 mg/m^3 as above.	No statistically significant differences in survival and weight gain or malignant lymphoma incidence				
			250 mg/kg	50 mg/kg		
Rats, Sprague-Dawley, ♂, ♀	Gavage/250 mg/kg 4-5 d/wk, 52 wks 50 mg/kg as above	Mammary Carcinomas	7/32	4/30	3/30	Maltoni and Scarnato (1979)
		Zymbal Gland Carcinomas	8/32[b]	2/30	0/30	
		Leukemias ♂	4/33	0/30	0/30	
		♀	1/32	2/30	1/30	
		Total Leukemias	5/65	2/60	1/60	
Mice, C57BL/6N[a]	Subcutaneous/0.9-2.6 g/kg b.w./injection 2x/wk, 54 wk (the average dose rate was approx. 750 mg/kg b.w./day)	Granulocytic Leukemia	8/45		3/36	Ward *et al.* (1975)
		Total Tumors	26/45		22/36	

TABLE 5-5. CARCINOGENICITY OF BENZENE IN EXPERIMENTAL ANIMALS (Continued)

Species	Route/Dosage	Results	Reference
Mice, C57BL6[a] AkR, C3H, DBA2 30 ♂	Subcutaneous/0.001 ml/wk one month of age until death (∿50 mg/kg/wk for lifetime)	Incidences in test mice of leukemia of aplastic anemia were not significantly different from control. (These strains were selected because of susceptibility to hematological problems.)	Amiel (1960)
Mice, C3H	Subcutaneous/∿50 mg/ once/wk. Presumed for 43 wk (∿7 mg/kg b.w./day for lifetime)	Leukemia incidence: 6/20[b] before 300 days in test group. 29/212 before 300 days in untreated controls.	Kirschbaum and Strong (1942)

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[a] These strains of mice are susceptible to induction of lymphomas.

[b] $p < 0.05$; chi-square test, one-tailed, comparison to controls.

Sufficient evidence demonstrates that benzene is a probable leukemogen in humans and probably causes other hematological disorders, especially pancytopenia. The dose-effect relationship between benzene exposure and the incidence of these diseases in humans is uncertain, primarily because of the absence of individual exposure data. Most importantly, mean exposure data for an entire work force may underestimate the exposure to individuals who develop the disease.

Further uncertainties are introduced when the occupational exposure levels are converted to a water concentration at which a lifetime consumption of 2 l/day will give the same total dose. Hattis and coworkers (1980) briefly discuss two reasons why it may be inappropriate to assume that all increments of exposure (by increased concentration or by increased duration) are equivalent. First, high-level occupational exposure could be far more effective in producing leukemia than low-level environmental exposure; thus, the effect of low-level exposure could be overestimated. Second, workers experienced their exposures as adults, whereas lifetime exposure means exposure to persons during childhood as well. If leukemia is a multistaged process and if benzene affects the early stages of that process, then the longer the time interval of exposure to benzene, the greater the risk of developing leukemia. Thus, the effect of lifetime exposure could be underestimated, when based on the dose conversion from the occupational data.

5.1.4.2 Additional Health Effects

Aside from the reported hematological effects of chronic benzene exposure, most adverse effects associated with benzene exposure are of an acute nature and occur at considerably higher exposures. High air concentrations of benzene can result in acute central nervous system effects ranging from mild euphoria, giddiness, staggering gait to paralysis, convulsions and potential death from respiratory arrest and/or cardiovascular collapse. Air concentrations in the vicinity of 64,000 mg/m^3 for 5-10 minutes are generally lethal.

Teratogenic effects have been observed in mice with very high exposures (3ml/kg on day 13 of gestation). Other toxic effects noted in pregnant rats and the developing embryo include decreased body weight in mothers, decreased litter size, embryonic resorptions and decreased fetal weights. These effects occurred with continuous inhalation exposure to benzene concentrations between 370 and 1783 mg/m^3. The U.S. EPA (1980) concluded that benzene is unlikely to be a potential teratogen.

5.1.5 Estimated Dose/Response Relationship for Cancer

Below, an estimate is derived for the potential lifetime carcinogenic risk to humans as a result of the ingestion or inhalation of benzene at a constant daily rate.

Ideally, this problem would be approached in two ways:

- Given human dose/response data (generally from retrospective studies of past occupational exposure, or of unusually high ambient exposure levels), various extrapolation models would be applied to obtain an approximate dose/response relationship (a relationship giving percent excess carcinogenic response as a function of daily dose or exposure level).
- Given dose/response data from controlled experiments on laboratory animals, the animal doses would be converted to estimated equivalent human doses, and again the various extrapolation models would be applied to obtain an approximate human dose/response relationship.

The advantage of the first approach is that the results are most relevant to humans because the "test" subjects are humans. Extrapolation of effect levels obtained from animal studies to "equivalent human doses" adds a degree (unquantifiable) of uncertainty to the dose/response relationship derived for man due to possible differences in susceptibility, pharmacokinetics, repair mechanisms, etc.

On the other hand, in retrospective human studies, the exposure levels, duration of exposure, and even response rates (carcinogenic responses per exposed population) are usually "best estimates." Furthermore, unknown factors (e.g., exposure to carcinogens other than the one in question) may seriously bias the data. Information on exposure, response, and general circumstances for the laboratory animals is accurate, because these are design parameters. Also, controlled animal experiments can yield a broader range of dose/response data points, which allows straightforward application of the extrapolation models. Usually this is not possible from human retrospective studies because of the insufficient data.

In addition to the uncertainties inherent in the type of data used in the analysis, other important and largely unquantifiable sources of uncertainty exist:

- The main purpose of risk analysis is to use observed response rates at relatively high exposure levels to extrapolate expected response rates (risks) at the relatively low levels that might be found in the environment. However, the extrapolation models cannot be tested at low exposure levels of concern (low enough to keep excess lifetime risk per capita around 10^{-5}). In that the mechanisms of carcinogenesis are not fully understood, there is no basis for choosing among a variety of different models. These models make similar risk predictions at high exposure levels, but markedly different predictions at low exposure levels. No attempt has been made to quantify the uncertainty inherent in the choice of an extrapolation model; rather, a variety of models has been applied to establish a range of potential risk. (No attempt has been made to determine statistical confidence bounds because of the unquantifiable uncertainties inherent in this analysis.)

- Test subjects, either humans or laboratory animals, are rarely exposed to any carcinogen for an entire lifetime. In the occupational setting, humans may be exposed 40hr/week for 5-10 years during the middle years of their lifetimes. Laboratory animals may be given doses daily, weekly, or only once; administration of the carcinogen rarely begins until the animals are reasonably mature, and usually ends some weeks before the animals are killed. The interest here is the potential risk as a result of lifetime exposure. Since the exposure period is significantly less than the lifetime of the test subjects, a simple linear extrapolation is performed to determine a lifetime dose equivalent to the actual dose. However, this extrapolation of intermittent or short duration exposure to equivalent lifetime exposure largely disregards such factors as recovery by normal repair mechanisms, clearance of body burdens, etc., during nontreatment periods.

- Peculiarities, such as the lack of control groups or contradictory results from equally valid studies, may make analysis difficult. After the elimination of irrelevant or highly questionable studies, this analysis was based on the study that yielded the most pessimistic results; thus, the analysis is conservative.

In summary, the potential lifetime carcinogenic risk to humans of a substance can be estimated by applying a variety of dose/response extrapolation models to human dose/response data and/or to human equivalent dose/response data based on laboratory animal data. Uncertainty arises in the estimation of human exposure and response, in the conversion of animal exposure to human equivalent exposure, and in the application of the dose/response extrapolation model themselves. Even greater uncertainty arises in the conversion of sporadic or short-term exposure to equivalent lifetime exposure. In any case, present scientific methods do not permit a more accurate or definitive assessment of lifetime human carcinogenic risk.

5.1.6 Discussion of Available Data

Although not ideal, the available human data for benzene are relatively good. The inherent validity of extrapolation within species far outweighs the quantitative uncertainty in the human data. The CAG (U.S. EPA 1978a) and MIT (Hattis et al. 1980) groups have done risk analyses based on the studies of Aksoy and coworkers (1977), Infante and coworkers (1977a, b) and Ott and coworkers (1978). Both CAG and MIT computed potential risks of lifetime exposure to benzene in air at low levels. The analysis below computes risk from ingestion of benzene. The study of Ott et al. (1978) was not used for these risk calculations. It is believed that their data are inconclusive because of the relatively few deaths evaluated (only 3 deaths due to leukemia), which tends to disproportionately affect risk estimates.

Having carefully considered both the CAG (U.S. EPA 1978a) and MIT (Hattis et al. 1980) analyses of risk, this discussion is limited to noteworthy deviations from these two analyses. The interested reader is urged to review these analyses to appreciate the sources of discrepancies, the uncertainties involved, and the rationale of the risk calculations. Differences in predicted risks stem from differences in exposure estimates, response estimates, and model equations. The first two categories are the input data and are given in Table 5-6 for the three analyses that were based on the Infante and Aksoy studies.

5.1.6.1 Infante Study

The CAG analysis considered total leukemias, 9 cases, in the exposed worker group vs. 1.25 in the nonexposed controls for a relative risk (R) of 7.2. The per capita probability of dying from all forms of leukemia at zero exposure to benzene [noted here as $P_L(0)$] was given by the CAG in their Table 2 as $P_1 = 0.0067$. This report concurs with the MIT group's choice to change R and consequently $P_L(0)$ to reflect consideration of only non-lymphatic leukemias. This makes a comparison with the analysis of the Aksoy data more appropriate and is consistent with the view that benzene exposure is associated much more closely with the non-lymphatic leukemias.

The occupational exposure concentration estimate by the CAG group for the Infante study is considered unrealistically low (see Section 5.1.3.2). Yet, the duration of exposure (25-36 years) may be unrealistically high as pointed out by the MIT group. The longer duration of exposure has been maintained to compensate for the likely underestimate in exposure concentration. Finally, the dose (see calculation Table 5-6) was computed on a mg/day basis assuming, as discussed in Section 5.1.3.2, that response is dependent on absorbed dose and is independent of exposure route.

TABLE 5-6. COMPARISON OF INPUT DATA FOR CALCULATION OF RISK OF LEUKEMIA FROM BENZENE EXPOSURE

		Input Data			
				Exposure	
Study	Analysis	R	$P_L(0)$[a]	Occupational	Lifetime Equivalent
Infante	CAG	7.2	0.0067	40-23 ppm x 25-36 yrs	2.8 ppm
	MIT	10.7	0.0045	40-23 ppm x 5-15 yrs	0.84 ppm
	ADL	10.7	0.0045	345 mg/day x 25-36 yrs	150 mg/day[b]
Aksoy	CAG	19.9	0.0045	63.6 ppm x 9.7 yrs	4.2 ppm
	MIT	19.9	0.0045	(78.8 - 32.4) ppm x 9.7 yrs	9.8 ppm
	ADL	19.9	0.0045	3250 mg/day x 9.7 yrs	450 mg/day[c]

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[a] Lifetime probability of death due to non-lymphatic leukemia at zero exposure (see text).

[b] $\frac{(40 + 23)}{2}$ ppm x 1.2 m^3/hr x 8 hr/day x 3.19 $\frac{mg/m^3}{ppm}$ x .5 x 5 day/7 day = 345 mg/day; 345 x $\frac{(25 + 36)\ yr/2}{70\ yr}$ = 150 mg/day.

[c] (180 ppm x 1.2 m^3/hr x 10 hr/day + 22.5 ppm x .7 m^3/hr x 14 hr/day) x 3.19 $\frac{mg/m^3}{ppm}$ x .5 x 6 day/7 day = 3250 mg/day; 3250 mg/day x $\frac{9.7\ yr}{70\ yr}$ = 450 mg/day.

5.1.6.2 Aksoy Study

All three risk analyses agree on the response input data R and $P_L(0)$. Discrepancies do exist in estimating exposure concentrations. The reason for the much lower estimate by the CAG begins with the assumption that average exposure concentration can be estimated by the geometric mean of the midpoint of two intervals (15-30 ppm for non-working hours and 150-210 ppm for work hours). The arithmetic mean would be more accurate since total exposure is proportional to the sum $C_1t_1 + C_2t_2 \dots C_nt_n$. Using (150+210)/2 for 10 hours and (15+30)/2 for 14 hours gives ≈88 ppm/hr compared to their geometric mean of 63.6 ppm. More importantly, the CAG reduces the average exposure again by adjusting for the 10-hour work day (i.e. 63.6 x 10/24 = 26.5 ppm); thus, it appears they adjust for lower non-work exposure concentrations twice. The MIT estimate is somewhat lower than the ADL estimate and stems from use of the geometric mean of a "worst" case and "best " case. MIT "worst" case is interpreted here as being closer to an average case since 180 ppm and 22.5 ppm are the arithmetic means of the CAG estimates, respectively, of the working concentration (150-210 ppm from CAG) and of the non-working concentration (15-30 ppm from CAG). Furthermore, their use of the geometric instead of the arithmetic mean results in a smaller exposure. The estimate given in Table 5-6 is more straightforward and believed to be more realistic.

In the conversion of occupational exposure data to lifetime average daily ingestion, the occupational exposure via inhalation is adjusted for 1.2 m^3/hr respiratory rate, 8- or 10-hour work days, a 3.19 mg/m^3 per ppm conversion factor, 50% inhalation retention, 5 (or 6) days/7-day work week, and average 70-year lifetime. These factors were utilized in Table 5-6. Tables 5-7 and 5-8 also provide a rough basis for comparing the predicted risks between studies by relating ppm to mg/day.

5.1.7 Application of Dose/Response Models to Estimation of Human Risk

The ADL risk predictions utilize two different models, the first model is the one-hit model (Arthur D. Little, Inc., 1980):

$$P(x) = 1 - e^{-(A+Bx)}$$

which is very closely approximated by the so-called linear model, utilized by CAG and MIT, for small values of P(x); that is for P(x) <.1,

$$P(x) \approxeq A+Bx$$

where P(x) is the lifetime probability of leukemia at dose x. For clarity, the notation $P_L(x)$ is used. The assumption here, as with CAG and MIT, is that "R", the relative risk of leukemia for benzene-

TABLE 5-7. PREDICTED EXCESS LIFETIME LEUKEMIAS PER MILLION POPULATION DUE TO BENZENE INGESTION (INHALATION), BASED ON THE STUDY OF INFANTE AND COWORKERS.

	Ingestion Rate in mg/day (Equivalent inhalation concentration in ppm)[a]						
	.01 (.00028)	.03 (.00084)	.1 (.0028)	.3 (.0084)	1 (.028)	3 (.084)	10 (.28)
ADL linear $B \cong .0003$ per mg/day	3	9	30	90	300	900	3000
ADL Log Probit $A \cong -3.9$	-	-	-	3	52	350	1800
CAG Linear $B \cong .01$ per ppm	2.8	8.4	28	84	280	840	2800
MIT Linear $B \cong .05$ per ppm	14	42	140	420	1400	4200	14000

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[a] $0.28 \text{ ppm} \times 3.19 \frac{\text{mg/m}^3}{\text{ppm}} \times 22.4 \frac{\text{m}^3}{\text{d}} \times 0.5 = 1 \text{ mg/day}.$

TABLE 5-8. PREDICTED EXCESS LIFETIME LEUKEMIAS PER MILLION POPULATION DUE TO BENZENE INGESTION (INHALATION), BASED ON THE STUDY OF AKSOY AND COWORKERS

	Ingestion Rate in mg/day (Equivalent inhalation concentration in ppm)[a]						
	.01 (.00028)	.03 (.00084)	.1 (.0028)	.3 (.0084)	1 (.028)	3 (.084)	10 (.28)
ADL Linear $B \cong 0.0002$ per mg/day	2	6	20	60	200	600	2000
ADL Log Probit $A \cong -4.0$	-	-	1	3	32	300	1400
CAG Linear $B \cong 0.02$ per ppm	5.6	17	56	168	560	1680	5600
MIT Linear $B \cong 0.009$	2.5	7.6	25	76	250	760	2500

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[a] $.028 \text{ ppm} \times 3.19 \frac{\text{mg/m}^3}{\text{ppm}} \times 22.4 \frac{\text{m}^3}{\text{d}} \times .5 = 1 \text{ mg/day}.$

exposed workers compared to age or time-matched control population, was independent of the length or age of exposure; that is,

$$R = \frac{P_o(x)}{P_o(0)} \text{ occupational } = \frac{P_L(x)}{P_L(0)} \text{ lifetime .}$$

Thus, $$R = \frac{A+B(x)}{A+B(0)}$$

where $$P_L(0) = A+B(0)$$

so that by algebraic manipulation,

$$B = P_L(0) (R-1)/x$$

where x is lifetime average daily exposure dose equivalent to the occupational exposure, $P_L(0)$ is the lifetime probability of leukemia with no or negligible benzene exposure, and B is the excess probability of leukemia per mg/day. Using the input data given in Table 5-6,

$$B_{Infante} = 0.00029 \text{ per mg/day}$$

$$B_{Askoy} = 0.00019 \text{ per mg/day}$$

Log Probit Model

$$P_E (x) = P_L(x) = \phi(A+\log_{10}x)$$

where $P_E(x)$ is the excess probability of leukemia at dose x and ϕ is the cumulative normal distribution. Using the same assumption about R as above [such that $P_L(0)R = P_L(x)$]; the following relationship is obtained:

$$P_E(x) = P_L(0) (R-1) = \phi(A+\log_{10} x)$$

and the values of the parameter A are as follows:

$$A_{Infante} = -3.89$$

$$A_{Askoy} = -4.02$$

Using the input data given in Table 5-6 and the two models, the predicted risks at various exposure levels were calculated for the two studies and are presented in Tables 5-7 and 5-8.

There is moderately good agreement among the eight separate predictions of risk at the higher levels of exposure (i.e. at >10 mg/day). An exception is the MIT estimate based on the Infante study, which is almost an order of magnitude higher than the others. This is because of a considerably lower estimated total exposure by the MIT group for this study. At lower exposure levels, five of the risk estimates are quite clearly parallel, the MIT/Infante prediction is consistently high, and the ADL Log Probit predictions for both the Infante and Aksoy studies are considerably lower because of the mathematical model employed.

5.2 HUMAN EXPOSURE

5.2.1 Introduction

Monitoring data on benzene in the environment indicate a wide range of benzene levels in the natural environment and in foodstuffs. The fate analyses also support the conclusion that benzene may occur in all environmental media. As discussed in the human effects section, it has been determined that benzene can be absorbed by all three routes of exposure--ingestion, inhalation, and dermal contact. The potential absorption of benzene by these three routes has been considered in the following analysis to estimate total daily absorbed doses.

Benzene concentrations in various media were estimated on a conservative basis in order to avoid underestimating the actual exposure that could occur. These data were combined with data on rates of air, water, and food intake and/or duration of exposure to estimate the amounts through each exposure route. Ideally, the absorption of benzene would be analyzed with respect to subpopulation factors such as age, weight, sex, breathing rates, food and water consumption, commuting and working patterns, etc. For benzene, such detailed data are not available and the variability and scarcity of the monitoring data do not justify a detailed analysis. Instead, in the analysis below, total daily absorption of benzene has been approximated for three broad population groups, based on their location with respect to major benzene sources of emission.

To illustrate that exposures to the general population are relatively low, occupational exposure to benzene has been evaluated for each exposure route (where appropriate) for a comparison with general population exposure groups.

5.2.1.1 Populations Exposed through Contaminated Drinking Water and Foodstuffs

The available data on benzene levels in drinking water are summarized in Section 4.3.2.1. These data indicate that benzene is detectable in drinking water from surface sources, generally, however at levels <2 μg/l. Maximum levels cited were <10 μg/l.

The U.S. EPA's Office of Drinking Water reports that approximately 100 million people, or about one-half the U.S. population receives surface waters in their homes. Thus, the remainder, who are usually people living in communities with less than 60,000 people, are receiving groundwater either from public or private wells. Although over 12 million private wells exist in the United States, the Office of Drinking Water did consider that working people often consume more water at their workplace than at home. Still, regardless of the kind of water supplied to their homes, most people are probably consuming both ground and surface waters on the average (Coniglio, personal communication 1980).

Data on levels of benzene in groundwater sources of drinking water are extremely sparse. Coniglio _et al_. (1980) found detectable levels of benzene in only 8.5% of ground supplies but in 21.6% of finished surface supplies. This may suggest that groundwater sources, on the average, are less likely to be contaminated with benzene than finished surface supplies.

Because of the high degree of uncertainty related to actual levels of benzene in ground water, an average level of 2 μg/l benzene in all drinking water supplies has been used to estimate the average exposure from all water sources. This level is considered conservative in that it more than likely overestimates the concentration in both surface and groundwater supplies. Given an average water consumption of 2 l/day, the daily intake of benzene in water is ∿4 μg/day.

Data on fluid consumption (see Appendix C) also suggest that this figure may be an overestimate. These data indicate that water is about 60% of total consumed liquids, while sodas, beer, juices, wines, spirits, etc. are the remainder. With the exception of water and juices, these liquids are typically ozonated or filtered through activated carbon to meet odor, appearance, and palatability criteria. Thus, the organics in the original water supply are removed. Therefore, using a standard water consumption of 2 l/day is likely to overestimate exposure to benzene by this route. Although great individual variability occurs in the consumption of food and drink, the 2-liter value probably leads to an overestimate of benzene absorption from drinking water.

Benzene may be ingested with contaminated foodstuffs. Reported concentrations in foods do not include all food groups, and it is unknown how representative these concentrations are of those in foods, in general. In the absence of other information, it was assumed that these data represent potential exposure levels. Estimates of total intakes for each food group consumed were taken from ICRP (1974). Also, included for comparison is the "conservative estimate" of 250 μg/day of benzene exposure through food (NCI 1977). For foodstuffs, an estimated benzene intake may be >30 μg/day and as much as 250 μg/day (NCI 1977).

Results from estimation of exposure to benzene through ingestion are documented in Table 5-9. Based on a water consumption of 2 l/day, at the concentrations shown in Table 5-9, the average benzene intake is 4 μg/day and a maximum of 20 μg/day. In order to avoid an underestimate in the true mean exposure through food ingestion, the NCI estimate of 250 μg/day was used to calculate the potential daily absorption of benzene from food.

TABLE 5-9. ESTIMATED BENZENE EXPOSURE THROUGH INGESTION

	Concentration[a] (μg/1)	Amount Consumed Daily[b] (1)	Daily Exposure (μg/day)
Water			
Median	2	2	4
High	10	2	20
Foodstuffs	μg/kg	kg	
Butter	0.5	0.056	0.03
Beef	2-19	0.025	0.004
Chicken, Lamb, Veal	< 10		
Eggs	500-1900	0.055	28-105
Haddock	100-200	0.026	2.6-5.2
Subtotal			31-108
NCI Estimate of Total Foodstuff Exposure[c]			250

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[a] The data are taken from Table 4-10.

[b] Data taken from ICRP (1974).

[c] Data taken from NCI (1977).

5.2.1.2 Populations Exposed through Inhalation

Sources of direct releases of benzene to the atmosphere include the plants that isolate benzene, the industrial plants using benzene, traffic, and gasoline distribution facilities.

Population groups exposed to benzene by inhalation have been categorized into four groups that are distinguished by atmospheric levels of benzene: urban (high levels due to traffic congestion), suburban (lower levels from less dense traffic), rural/remote (low levels--sparsely distributed vehicular sources), and user/manufacture sites (high level, point sources). These groups coincide with typically available monitoring data for atmosphere concentrations resulting from the broad range of emission categories. Cigarette smoking has been treated as a separate exposure situation.

The labor force in the vicinity of a source may be exposed 8 hr/day, while residents in the area of a source may be exposed up to 24 hr/day. In the latter case, emissions may be reduced or eliminated at the close of the working day, as a function of local meteorological conditions; and nighttime exposure could drop to the local background level. Thus, pollutant concentrations, which depend on the dispersion of emissions, will vary over time in any given location, even if the emission rate is absolutely constant.

Without performing site-specific modeling to determine actual concentrations, durations, and hence exposures, the analysis was simplified to the consideration of average and maximum observed concentrations. Because of the intermittent nature of point sources, the maximum concentrations are unlikely to exist longer than 8 hr/day and are probably much shorter in duration. The mean concentrations, which were obtained from monitoring data near sources, were applied to 24 hr/day exposure scenarios.

Cigarette smoking has also been determined to add to the amount of benzene inhaled and increase levels in the surrounding air. According to Drill and Thomas (1978), the average benzene exposure is 90 μg/cigarette. Based on data from the 1979 report from the U.S. Surgeon General, the average smoker (1.56 packs/day) would be exposed to 2.8 mg/day and retain 1.4 mg/day (Richmond 1981). The U.S. Surgeon General also reports a total of 54 million smokers in the United States in 1978 for all age groups.

Young _et_ _al_. (1978) have stated that "unknowing inhalation" in the home can occur from the use of paint strippers, carburetor cleaners. denatured alcohol, rubber cement, and arts and crafts supplies. These sources have not been documented, and exposures are assumed to be infrequent as well as dilute.

It has been assumed that the general population visits gas stations periodically, although the frequency ranges from perhaps once a day to once or twice per month. A frequency of one visit per week for a 10-minute duration was chosen as a representative pattern, which is equivalent to 0.02 hr/day. The assumption that a 10-minute/week exposure is equivalent to 0.02 hr/day is consistent with the guideline set by the U.S. EPA (U.S. EPA 1979), which states that lifetime carcinogenic risk is dependent upon total exposure, and not the frequency or duration of individual exposures. The units of minutes/week were converted to hr/day in order to conform to the units of exposures from other sources. In any event, the exposure due to gas station use by the general population is relatively small and does not play a crucial role in the risk assessment.

Occupational exposures to benzene by inhalation are analyzed at the OSHA standard and over a range of observed workplace values to provide contrast with ambient exposure scenarios. The standard established by OSHA is 10 ppm (32 mg/m^3) as a time-weighted-average for the 8-hour work day. Inhalation of benzene at this concentration would permit an absorption of 153 mg/day.

The product of the benzene concentration, duration of exposure, and appropriate respiratory rates were used to estimate potential daily exposure (see Table 5-10). Exposures were calculated using the average active adult breathing rate of 1.2 m^3/hr (16 hours), which falls to 0.4 m^3/hr during sleep (8 hours) (ICRP 1975). The numbers presented in Table 5-10 represent possible exposures to benzene, and include a respiratory retention factor of 0.5.

The results of the exposure calculations in mg/day (Table 5-10) show that nonoccupational inhalation intakes may range from 0.005-10 mg/day, while exposure at the occupational standard is 153 mg/day. The average exposure of residents near a refinery, which appears to be the source of the highest mean exposure (0.5 mg/day), is about 300 times lower than the exposure of workers at the OSHA standard of 10 ppm (31,920 µg/m^3). Other nonoccupational activities are associated with even lower relative exposures.

Urban and suburban areas do not differ greatly for typical benzene concentration values; however, urban areas have a larger and higher range. Average benzene concentrations in remote areas are only 1/5 and 1/3 the urban and suburban levels, respectively.

5.2.1.3 Percutaneous Exposure

Pure benzene is no longer readily available for residential use. The majority of solvents, paint removers, paints and other substances used in the home would contain only small amounts of benzene as a contaminant or possibly as a deliberately included component. As described in Section 5.1, the rate of dermal absorption for benzene is about 550 mg/m^2/hr. Assuming a situation involving a 5% benzene solution and a 1/2-hour exposure duration, the calculation is:

TABLE 5-10. ESTIMATED BENZENE EXPOSURE THROUGH INHALATION

Exposure Activity	Benzene Concentration Mean ($\mu g/m^3$)	Benzene Concentration Range[a] ($\mu g/m^3$)	Exposure Duration (hr/day)[b]	Exposure[c] Mean (mg/day)	Exposure[c] Range[a] (mg/day)
Nonoccupational Activities in					
Urban Areas	8.0	0.5-412	≤ 24	0.09	0.005-4.6
Suburban Areas	5.1	3.2-8.1	≤ 24	0.05	0.04-0.09
Rural/Remote Areas	2.2	1.0-3.5	≤ 24	0.03	0.01-0.04
Near Manufacture /User Sites					
Chemical	20	1-111	8	0.01	0.005-0.55
Refinery	46	3-824	≤ 24	0.5	0.04-10
Gas Station Use	860	100-5400	0.02	0.01	0.001-0.08
Cigarette Smoking	90 μg/cigarette		1.6 packs	1.4	-
Occupational Activities					
Outdoor In-traffic Job	12.3	5.9-21.3	8	0.06	0.03-0.1
In Benzene Recovery Plant	4200	2700-10,700	8	20	13-51.5
Range of Known Industrial Levels	?	0-483,300	8	?	0-2320
Gas Station Employees	260	110-5400	8	1.2	0.5-26
Exposure at the OSHA Standard	31,920	--	8	153	--

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[a] The range given represents the spread of available data and is not meant to imply absolute limits.

[b] The symbol $\leq$ indicates that because of the nonconstant character of the emissions, the exposure at the levels shown probably does not occur over the entire day.

[c] Exposures were calculated based on a respiratory retention factor of 0.5.

$$550\ \mathrm{mg/m^2/hr} \times 0.072\ \mathrm{m^2} \times 0.05 \times 0.5\ \mathrm{hr} = 1\ \mathrm{mg}.$$

It is not realistic to assume that this level of exposure is typical; however, for some small populations, it may represent a sporadic exposure.

For comparison, assuming a worst case exposure in an industrial situation in which an employee had both hands immersed in benzene for 1 hr/day, the resulting exposure would be:

$$550\ \mathrm{mg/m^2/hr} \times 0.072\ \mathrm{m^2} \times 1\ \mathrm{hr/day} = 40\ \mathrm{mg/day}$$

where 0.072 m^2 is the surface area of the hands. In Section 5.1.1.1, a permeability factor was estimated to compute absorption of benzene vapor through the skin; this factor was 0.002 $m^3/(m^2 \times hr)$. At the OSHA standard of 10 ppm (32 mg/m^3), absorption of benzene vapor into the body via the skin would be $\sim$0.9 mg/8-hour work day.

5.2.2 Comprehensive Exposure Scenarios

The results of the exposure estimates are summarized in Table 5-11. These data have been used in comprehensive exposure scenarios for all routes (see Table 5-12). Scenario A involves the potential exposure of urban dwellers (149,639,720 people or 74% of the 1970 Census population), and would include exposure by inhalation, ingestion of predominantly surface water (110 million people drink surface water supplies, which are usually supplied to urban areas whose size is greater than 60,000 people), food consumption, and the use of gas stations. Percutaneous exposure was not included in this comprehensive scenario because it was assumed to be restricted to a small subpopulation using benzene sporadically. The total typical daily exposure is about 0.4 mg/day. Cigarette smoking could add 1.4 mg/day to this amount, as well as to the amounts in the scenarios described below.

Considering the next largest population, rural dwellers (53,572,206 people as of 1970 or 26%), as Scenario B, inhalation exposure was included as well as foodstuffs, drinking water, and gas station usage. Although the drinking water supply is nearly 100% from groundwater, average exposure levels are unknown. For the purpose of calculation, therefore, the same value, i.e., urban value, was used. The total exposure for the rural scenario is approximately 0.3 mg/day.

Scenario C, involves residents near a user or manufacturing site. In this case, inhalation dominates the other routes by adding up to 0.5 mg/day for a total of 0.8 mg/day. The number of people involved in this scenario cannot be accurately determined at this point; however, it is likely to be small, compared with Scenarios A and B.

TABLE 5-11. SUMMARY OF ESTIMATED BENZENE EXPOSURE AND ROUTES

Route and Activity	Mean Daily Intake (mg/day)	Estimated Exposure Population (millions)[b]
Ingestion		
Water	0.004	220
Food[a]	0.250	
Inhalation -- Nonoccupational		
Urban	0.1	150
Suburban	0.05	70
Rural	0.03	
Near Emission Sources	0.01-0.05	unable to estimate
Gas Station Use	0.01	220
Cigarette Smoking	1.4	54 million (1978)
Inhalation -- Occupational		
Outdoor In-traffic Jobs	0.05	unable to estimate
Industrial	20	unable to estimate
Gas Station Employees	1.5	unable to estimate
At Occupational Standard	153	unable to estimate
	Worst Case (mg/day)	
Percutaneous		
Occupational -- Liquid	40	an undeterminate subset of 0.1 million
Occupational -- Vapor	0.9	
Residential -- Liquid	<1.0	subpopulation unknown but quite small

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[a] There are as yet insufficient data to determine truly typical values. These data are the NCI's (1977) "conservative estimate."

[b] Populations based on 1970 Census Data (U.S. Bureau of the Census 1979).

TABLE 5-12. COMPREHENSIVE EXPOSURE SCENARIOS FOR BENZENE

Route	Exposure by Scenario in mg/day		
	A	B	C
% of 1970 Population	74	26	?
Ingestion			
Water	0.004	0.004	0.004
Food	0.25	0.25	0.25
Inhalation			
Baseline	0.1 (urban)	0.03 (rural)	0.5 (near sources)
Gas Stations	0.01	0.01	0.01
Cigarettes[a]	1.4	1.4	1.4
Percutaneous			
Residential	<1.0[b]	<1.0[b]	<1.0[b]
TOTALS	0.4	0.3	0.8
Potential Maximum[c]	1.8	1.7	2.2

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[a] In 1978, a population of 54 million individuals smoked cigarettes.

[b] This amount not included in total exposure.

[c] These amounts include smoking.

Source: Arthur D. Little, Inc.

To provide a contrast with these three ambient exposure scenarios, potential industrial exposures of employees producing and utilizing benzene were calculated. If exposure occurs at the OSHA standard, the employee can add 153 mg/day to baseline (food and water and nonoccupational inhalation) exposure. If percutaneous exposure also occurs, an additional exposure to 40 mg/day is possible. It is possible that a very small number of individuals in this category (i.e., a most improbable event) will receive the maximum possible exposures from all routes, about 190 mg/day. Not only is the level of exposure unlikely, it would only occur for less than a lifetime duration. Nevertheless, these calculations indicate that occupational exposures to benzene are potentially much higher than nonoccupational exposures.

5.2.3 Summary

In comparison to the potential occupational exposure to benzene at the OSHA standard, the nonoccupational exposures are low. The total absorbed dose, excluding smoking, is on the order of 0.3-0.8 mg/day. At the average rate of 1.56 packs/day, smoking was estimated to add 1.4 mg/day to the total absorbed daily dose. In contrast, the contribution of water ingestion to total benzene absorption appears to be quite low; however, the contribution of food may be one-half the total for nonsmokers. Although the data on benzene levels in water and foodstuffs are scarce, the reported levels are considered to be indicative of the approximate benzene levels commonly found in these sources.

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6.0 BIOTIC EFFECTS AND EXPOSURE

6.1 EFFECTS ON BIOTA

6.1.1 Introduction

This chapter provides information on the exposure levels of benzene that cause mortality or disrupt physiologic functions and processes in aquatic organisms. Fairly extensive recent data exist for both marine and freshwater organisms, including fish (adult, juvenile, larval, and egg stages) invertebrates, plankton, algae, and microorganisms. The toxic effects of benzene have been studied on cells, tissues, organisms, and behavioral functions, such as reproduction, feeding, and locomotion. Basically, it appears that benzene disrupts the cell membrane permeability, which changes the ionic content of the blood and tissues, resulting in internal poisoning.

Primarily, static bioassay techniques have been used to test the effects of benzene on aquatic organisms. The static bioassay test utilizes one initial exposure to an appropirate concentration of a chemical to determine toxicity. In flow-through bioassay tests, a fresh solution containing the test substance is continuously or periodically supplied to the organisms throughout the test period. Benzene is a highly volatile compound, only slightly soluble in water. The half-life in water (the time required for the concentration of a compound to drop to one-half of its initial value) is very short for benzene, approximately 4.5 hours because of evaporation (Buikema and Hendricks 1980). The problem of evaporation is inherent to both static and flow-through bioassay tests where concentrations are determined nominally, i.e., through introducing a measured amount of the substance other than direct periodic measurement during the bioassay. As a result, the validity of the data from toxicity tests for volatile substances where the test solution is open to the environment is questionable (Buikema and Hendricks 1980).

No data on the toxicity of benzene to terrestrial biota were available.

6.1.2 Mechanisms of Toxicity

Several authors note that the basic mode of action of benzene, a fat-soluble anesthetic, appears to be the disruption of cell membrane permeability and changes in the ionic content of the blood and tissues. Though the mechanisms of toxicity are unclear, it has been noted that benzene causes an increase in cell permeability.

The mode of action may be disruption of the lipo-protein linkages of the membrane. Based on changes in the blood chemistry of young coho salmon, Morrow et al. (1975) suggest that narcosis resulting from changes in gill permeability causes ionic imbalance and internal CO_2 poisoning. Shifts in ionic balance would interfere with the fish's ability to control the gas content in the swim bladder, which may account for the observed loss of equilibrium. It is believed that benzene is absorbed across the fish gill directly into the blood. From there, it is transported to tissues, such as liver, muscle, and kidney where it may be oxidized to phenol (Brocksen and Bailey 1973).

Benzene may accummulate in the lipid-rich nervous tissue and result in narcosis. In turn, narcosis may cause respiratory depression and collapse by depressing the central nervous system (CNS). Depression of the CNS function will occur if the cells cannot maintain their proper ionic balance for nerve impulse transmission. At high concentrations of benzene, fish pass sequentially through phases of restlessness (rapid, violent, and erratic swimming), "coughing" or backflushing of water over the gills, increased irritability, loss of equilibrium, paralysis, and death (Leibmann 1960, Morrow et al. 1975).

Benzene can also cause acute anemia and decrease the oxygen transport capacity of the blood, which results in anoxia. The actions of benzene on the cell membrane, however, are rapidly reversible when the benzene stress is removed (Brocksen and Bailey 1973, Goldacre 1968, Morrow et al. 1975).

6.1.3 Freshwater Organisms

Toxicity studies on freshwater biota included tests on algae, three invertebrate species, and ten fish species (Tables 6-1 through 6-4). Because of the extreme volatility of benzene and poor static toxicity methodologies, no meaningful LC_{50} values for freshwater fish were obtained. In addition, because benzene solubility decreases as salinity increases, freshwater toxicity values should not be based on data obtained from marine organisms (Berry and Brammer 1977).

Other studies (U.S. EPA 1978a) report LC_{50} values for freshwater organisms similar to those reported in Buikema and Hendricks (1980). The range of LC_{50} values for five species of freshwater fish was 20.0 mg/l for the blue gill (*Lepomis macrochirus*) to 386.0 mg/l for the mosquito fish (*Gambusia affinis*) (Table 6-3).

Various authors (Buikema and Hendricks 1980) studied five algae genera under static conditions. Toxic effects varied from 0.001 to ≥1000 mg/l benzene, and Dunstan et al. (1975) state that 10 mg/l benzene appeared to be the inhibition threshold for all marine algae tested except for the green alga *Dunaliella*. This alga was capable of good growth up to 100 mg/l benzene. It was concluded that benzene concentrations would rarely be as high as 10 mg/l except for extremely short periods because of its volatility (Dunstan et al. 1975).

TABLE 6-1. EFFECT OF BENZENE ON AQUATIC FLORA

Species	Toxicant Concentration (mg/1)	Length of Experiment	Effect	Reference
ALGAE				
Amphidinium cartarae	0.001 - 100	3 days	inhibited growth	Dunstan et al. (1975)
Skeletonema costatum	0.001 - 50	3 days	no effect	Dunstan et al. (1975)
	100	3 days	inhibited growth	Dunstan et al. (1975)
Dunaliella tertiolecta	0.001	3 days	stimulated growth	Dunstan et al. (1975)
	0.01 - 100	3 days	no effect	Dunstan et al. (1975)
Cricosphaera carterae	0.001 - 20	3 days	no effect	Dunstan et al. (1975)
	50 - 100	3 days	inhibited growth	Dunstan et al. (1975)
Skeletonema costatum	0.1 - 10	10 days	no effect	Atkinson et al. (1977)
	20 - 100	10 days	inhibited growth	Atkinson et al. (1977)
Chlorella vulgaris	25 - 500	10 days	no effect	Hutchinson et al. (1972)
	1000 - 1744	10 days	inhibited growth	Hutchinson et al. (1972)
Chlorella sp.	55 - 553	12 hours	reduced photosynthesis/ respiration ratio	Potera (1975)
FUNGI				
Saccharomyces anomalus	312		no effect	Levan (1947)
	625 - 937		toxic	Levan (1947)
	1016 - 1250		lethal	Levan (1947)
PLANTS				
Anacharis canodensis (Elodea)	741	1 hour	killed plants	Currier and Peoples (1954)

TABLE 6-2. ACUTE TOXICITY OF BENZENE TO INVERTEBRATES

Species	Habitat	Test Duration (hour)	Test Type	EC$_{50}$ or LC$_{50}$ (mg/l)	Reference
Tigriopus californicus	SW	168	S	>0.087	Barnett and Kontogiannis (1975)
Crassostrea gigas (larvae)	SW	48	S	0.38	LeGore (1974)
Balanus amphitrite	SW	1	S	>1.0	Barash (1974) Hubault (1936)
Aedes aegypti	FW	24	S	≃1.4	Berry and Brammer (1977)
Brachionus plicatilis	SW	48	S	>1.5	Eldridge and Echeveiria (1977)
Crago franciscorum	SW	24	S	22.0	Benville and Korn (1976)
Palaemonetes pugio	SW	48	?	33.0	Tatem and Anderson (1974)
			S		Potera (1975)
		24	S	43.5	Tatem and Anderson (1974)
		48	S	35.0	Tatem and Anderson (1974)
		96	S	27.0	Neff *et al.* (1976) Tatem and Anderson (1974)
(larvae)		74	S	74.4-90.8	Potera (1975)
(adult)		74	S	37.5-38.0	Potera (1975)
Artemia salina	SW	24	S	66.0	Price *et al*. (1974)
Nitrocra spinipes	SW	24	S	82-111.5	Potera (1975)
Daphnia magna	FW	48	S	203.0	U.S. EPA (1978a)
Cancer magister	SW	48	CF	>347.0	Caldwell *et al*. (1976)
		96	CF	108.0	Caldwell *et al*. (1976)
		240	CF	<5.5	Caldwell *et al*. (1976)

TABLE 6-3. TOXICITY OF BENZENE TO FRESHWATER ORGANISMS[a]

Species	Concentration (mg/l)	Effect
Bluegill sunfish (*Lepomis macrochirus*)	20.0	48-hr LC_{50}
Bluegill sunfish (*Lepomis macrochirus*)	22.49	96-hr LC_{50}
Fathead minnow (Pimephales promelas)	32.0-33.7	"
Goldfish (*Carassius auratus*)	34.42	"
Guppy (*Poecilia reticulatus*)	36.6	"
Mosquitofish (*Gambusia affinis*)	386.0	"
Daphnia magna	203.0	48-hr LC_{50}
Daphnia magna[b]	356-620	"
Daphnia pulex[b]	265-345	"
Daphnia culcullata[b]	356-390	"
Daphnia magna	>96.0	Chronic value
Alga (*Chlorella vulgaris*)	525.0	48-hr EC_{50} Reduction in cell no.

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[a] Data taken from Table 6-1 through 6-4, U.S. EPA (1978a), except where noted.

[b] Cntrow and Adema (1978).

TABLE 6-4. TOXICITY OF BENZENE TO SALTWATER ORGANISMS

Species	Test Duration (hour)	Test Type	LC_{50} value (mg/l)	Reference
Striped bass (*Morone saxatilis*)	72, 96	CF	9.6	Meyeroff (1975)
		S	15	Brocksen and Bailey (1973)
	24	S	6.9	Benville and Korn (1977)
	96	S	5.6	Benville and Korn (1977)
Anchovey (*Engraulis mordax*)	48	SR	4-55	Struhsaker *et al.* (1974)
Pacific herring (*Clupea pallasi*)	48	SR	17.6-22	Struhsaker *et al.* (1974)
Coho salmon (*Oncorhynchus kisutch*)	24	S(?)	50	Morrow *et al.* (1975)

Three species of daphnids tested for sensitivity to benzene were generally more tolerant than fish. LC_{50} values ranges from 203.0 to 620 mg/l, both for Daphnia magna. A chronic value of 96.0 mg/l for Dahpnia magna was reported by the U.S. EPA (1978b).

6.1.4 Marine Organisms

The toxicity data indicate that the concentrations of benzene causing acute toxicity to most invertebrates are higher than the concentrations normally measured in the environment. Acute toxicity data for nine saltwater species are presented in Table 6-4.

Buikema and Hendricks (1980) cite the only meaningful LC_{50} value for saltwater fish, i.e., Morone saxatilis (striped bass), which was obtained from a continuous flow bioassay (Table 6-4). Other data on various marine species indicate that acute toxicity values range from 5.6 mg/l for striped bass to 22.0 mg/l (U.S. EPA 1978a) for larvae of Pacific herring and 55 mg/l for the anchovy (Engraulis mordax).

Sublethal Effects

Various sublethal effects of benzene on aquatic organisms have been studied, including enhancement or depression of growth and development, oxygen consumption, and feeding behavior. A significant number of female Pacific herring spawned permaturely when exposed to 0.1 and 0.8 mg/l benzene (Struhsaker 1977). Benzene was rapidly accummulated in the ovaries and spawned eggs. Dead eggs were also observed in the ovary. Herring eggs exposed to an initial concentration of 45 mg/l benzene took longer to develop. Some development abnormalities occurred in larvae exposed to 45 mg/l benzene. The LC_{50} for early Pacific herring larvae exposed to benzene was 20-25 mg/l. Larvae were more sensitive than eggs to benzene; however, the larvae had a greater capacity to recover from benzene stress (Struhsaker et al. 1974).

As benzene concentration increased, the wet weight, dry weight, and fat content of juvenile striped bass decreased. This effect may have resulted from impaired feeding, documented at higher concentrations. The energy to metabolize benzene could also decrease efficient utilization of energy for growth and fat deposition (Korn et al. 1977).

Studies have noted that Pacific herring larvae exposed to 6.7 and 12.1 mg/l benzene have exhibited reduced feeding movements. Struhsaker and coworkers (1974) substantiated this effect by the lower incidence of fish larvae with food in their guts. Juvenile striped bass acutely exposed to an average benzene concentration of 6.0 mg/l were unable to locate and consume their food ration. After 1 week, feeding success improved, and by the end of 4 weeks, striped bass exposed to 3.5 mg/l consumed 50% of their ration (Korn et al. 1977).

Several studies cited in Buikema and Hendricks (1980) indicate that fishes exposed to sublethal doses of benzene exhibit significant changes in oxygen consumption, and that the effects vary with life stage. Studies on Pacific herring, chinook salmon and striped bass indicate that oxygen consumption generally increases with exposure to greater concentrations of benzene. There are several theories on the mechanism by which oxygen consumption increases. One is that it results from the oxidation of benzene to phenol by body tissues (Brocksen and Bailey 1973).

6.1.5 Factors Affecting the Toxicity of Benzene

Certain environmental conditions may affect the results of toxicity tests, both in the field and the laboratory. One principal parameter that may affect the toxicity of benzene is salinity. The resistance of copepods increases as salinity increases. However, a reverse response has been noted for larval grass shrimp, while the adults were not as salinity dependent (Potera 1975). These differences in response may reflect the lower solubility and thus biological availability of benzene in saltwater (Lee *et al.* 1974) and differences in organism size between the two age classes thus influencing uptake.

Temperature interactions have been studied only for algae, harpacticoid copepods, and grass shrimp. Adult grass shrimp were more tolerant at lower test temperatures. This suggests that benzene enters the organism more slowly perhaps because of lower metabolic rate (Potera 1975).

The factor of size as an effect on the response of fish to benzene has been investigated and related to gill surface area. Less area is related to less accumulation and excretion over time. Brocksen and Bailey (1973) have also speculated that the different susceptibilities of the species tested may be related to differences in lipid-rich tissue and the biochemical pathways associated with fat metabolism. In addition, several studies in Buikema and Hendricks (1980) indicate that the sensitivity of an organism varies with the life stage tested. Larger and/or more mature organisms are generally more resistant to benzene. However, Struhsaker (1977) found that the eggs of the northern anchovy and the Pacific herring were more resistant than the early larvae.

6.1.6 Conclusions

According to the literature surveyed, the lowest concentration of benzene at which effects have been observed in aquatic organisms is 0.001 mg/l. This concentration affected growth in several algae species. Acute effects on freshwater plants (*Elodea*) were found at 741 mg/l. Toxic effects on algae were noted in concentrations ranging from 0.001 to 1000 mg/l benzene; the alga *Chlorella vulgaris* was not affected in concentrations of <500 mg/l benzene. Acute effects for invertebrates ranged from >0.087 mg/l benzene for the copepod *Tigriopus californicus* to 347.0 mg/l benzene for the crab Cancer magister. Daphnids were the most resistant of the freshwater invertebrates, with LC_{50} values ranging from 203 to 620 mg/l for four species tested. Some of the available laboratory data for freshwater fish are questionable because of the static bioassay methodology; however, other studies report LC_{50} values ranging from 20.0 mg/l for the bluegill sunfish (Lepomis macrochirus) to 36.6 mg/l for the guppy (Poecilia reticulatus). The least sensitive fish in this study was the Mosquito fish (Gambusia affinis) with a LC_{50} of 386.0 mg/l.

Marine toxicity data on adult fish were limited. The only value available, an LC_{50} of 5.6-10 mg/l benzene, was for striped bass (Morone saxatilis). An overview of the data suggests that life cycle is an important factor in the sensitivity of a species to benzene. From tests on several marine fish species, in general, larvae were found to be more sensitive than eggs to benzene; however, larvae had a greater capacity to recover from benzene.

Various concentrations of benzene have been shown to cause sublethal effects on organisms, including changes in feeding, growth and development, locomotion, and oxygen consumption. Premature spawning in Pacific herring was noted at 0.5 mg/l benzene; concentrations of 6.7 and 12.1 mg/l benzene reduced feeding activity in the same species. Several environmental factors influence the toxicity of benzene; the major parameters are temperature and salinity. How these parameters affect toxicity varies among organisms. Other than the decrease of benzene solubility with the increase of salinity, no definitive conclusion can be drawn regarding the effects of these parameters on the toxicity of benzene.

In summary, laboratory tests can establish general concentration ranges that demonstrate certain effects. However, these ranges are not rigidly defined, and may overlap as a result of differences among species, life stages or environmental variables. Test ranges include:

• $\geq$ 0.001 mg/l.	The lowest concentration at which toxic effects were served in any aquatic organisms.
• 0.001-20.	Concentrations caused acute and sublethal effects on adult fish including, striped bass, Pacific herring, and trout (Salmo gardnerii). This range also affected behavior and physiologic processes in Pacific herring larvae and juvenile striped bass.
• 20-36 mg/l.	Range reported acutely toxic to several (primarily sensitive) freshwater fish.

• 36 - 100 mg/l.	Concentrations in this range inhibited growth in several species of freshwater algae; and in one species, it stimulated growth. These concentrations were acutely toxic to several small invertebrates including copepods.
• 100 - 400 mg/l.	Concentrations had chronic and acutely toxic effects on a variety of organisms, including the resistant mosquito fish, Dungeness crab (*Cancer magister*), and several Daphnids.
• 400 - 1744 mg/l.	Such concentrations inhibited growth, and reduced photosynthesis--respiration ratio in the resistant algae species, *Chlorella*.

6.2 EXPOSURE OF BIOTA TO BENZENE

Benzene is a fairly common substance in aquatic systems in the United States, and has been detected in numerous types of waters, including drinking water, rivers, chemical plant effluents, well water, and lakes (Buikema and Hendricks 1980).

Industrial operations, which are the main sources of direct aquatic contamination, include: chemical production and processing, coating operations, and storage and transportation. Direct input to the environment can occur via spills, leaks, and/or effluents from industrial sites. The losses from production are concentrated primarily along the Texas Gulf coast and in the Northeast (Buikema and Hendricks 1980).

Aquatic exposures can occur in any water contaminated with benzene from a discharge, runoff, or as the result of an intermedia transfer from land (landfill leachate infiltration of ground and the surface waters) or air (rainout).

Our analysis will discuss probable levels of benzene involved in aquatic exposures and compare them with the concentrations known to have acute toxic or sublethal effects.

6.2.1 Exposure Route

No information addressed the ingestion of benzene by aquatic biota. The available data suggest that the primary mechanism of toxicity of benzene to fish is changes in gill permeability, which results in internal CO_2 poisoning. In chronic bioassays, benzene was found to impair feeding in juvenile striped bass (*Morone saxatilis*); however, this effect was not necessarily attributed to the ingestion of benzene.

6.2.2 Fish Kills

No data were found in the literature concerning any fish kills related to benzene in aquatic environments.

6.2.3 Monitoring Data

Water quality data provided by STORET and Battelle (1979) (see Chapter 4), indicate that ambient water concentrations of benzene are generally quite low; 76% of 185 sampling stations reported values <10 μg/l. Overall, effluent levels of benzene are not higher; 93% of 274 effluent monitoring stations also reported values of ≤10 μg/l. Some localized areas have concentrations of benzene; however, these areas are located in Missouri, Kansas, and Michigan. These higher concentrations (80-639 μg/l), which were up to seven times greater than those in other areas, were in the vicinity of chemical plants and petroleum refineries. Both ambient and effluent data were gathered near chemical plants; mean effluent concentrations were 50 times greater than mean ambient concentrations. No data were given, however, on the distance downstream or the length of time these effluent concentrations persisted. In Connecticut, Florida, and Missouri, concentrations near several chemical companies have reached 1000 μg/l.

6.2.4 Exposure

The monitoring data for benzene are not extensive, thus it is difficult to present a comprehensive assessment of exposure levels of benzene in aquatic systems. Based on the data available, however, where benzene is detected it is almost always found in low (μg/l) concentrations. These levels are generally lower than concentrations that have been determined to be toxic to aquatic biota by a factor of 2000. The few incidences of higher concentrations associated with chemical plants are still below (by 100x) those levels found to be toxic to aquatic organisms. No data were found on the temporal or areal extent of these concentrations. Based on the output from EXAMS, estimates were made of the downstream distances that slow elevated concentrations of benzene. A river with dimensions of those in EXAMS will show 99% depletion of benzene concentrations within ∿60 miles of the discharge point (see Section 4.4.3.2). Note that river characteristics vary greatly, especially in regard to flow, and to a lesser extent, depth and velocity. Whether an effluent results in a hazardous level of benzene downstream depends primarily on the upstream flow available for dilution and on the strength of the effluent. Depth and stream velocity also affect the rate of attenuation.

The river velocity used in EXAMS is typical in streams of many different widths and depths, while streams with a similar cross section of the EXAMS model may have significantly different water velocities. These factors will have a bearing on actual concentrations of benzene, which will occur in the water from a discharge. Large sluggish streams could have higher concentrations of benzene over a smaller area. On the other hand, large streams, because of their deep channels may only appear sluggish and may instead carry the benzene far downstream, while shallow, more turbulent streams will dissipate concentrations within a short distance.

The EXAMS model may underestimate the actual water column concentrations because it allows complete mixing throughout the 1000-meter x 100-meter x 3-meter river segment. Many discharges will form effluent plumes, which will remain distinct from the flow of the river and will naturally contain higher concentrations than EXAMS would predict for the entire segment. Although the EXAMS results indicate that only the mildest effects (inhibited growth to one alga species) may occur from exposure to the largest aquatic discharge modeled, more serious effects could be possible.

These considerations, however, when tempered with STORET data for ambient levels do not give rise to predictions of aquatic exposures at levels of benzene of concern for acute or serious chronic (sublethal) effects.

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7.0 RISK ESTIMATES FOR BENZENE EXPOSURE

7.1 HUMANS

Sufficient evidence demonstrates a relationship between chronic human exposure to rather high benzene concentrations (300-1200 mg/m^3) and hematological disorders, most notably leukemia. Aside from hematotoxic effects, other adverse health effects associated with benzene exposure are of an acute nature and occur at considerable higher exposure levels than ambient water and/or air concentrations of benzene.

Estimates of the potential lifetime risk of cancer from exposure to benzene are based on the modeling performed in Chapter 5. The results of four risk models that were utilized for benzene were presented in Tables 5-7 and 5-8 for two separate data sets. The results of coupling these results with the exposure scenarios presented in Table 5-11 are shown in Table 7-1 for the two data sets and the three comprehensive exposure scenarios, with smoking analyzed separately. Note that the following estimates of risk are based on typical, total benzene intakes for three very general exposure scenarios. Intakes were calculated for an average 70-kilogram human utilizing an inhalation retention factor of 0.5. The following risk estimates also assume that risk is independent of exposure route and assume no threshold for induction of carcinogenic effects.

Scenario A encompasses about 150 million urban dwellers or 74% of the 1970, U.S. population. Exposure might occur by inhalation, ingestion of drinking water (mostly from surface supplies), food consumption, and gas station usage. For this population, the average, daily exposure to benzene was estimated at 0.4 mg/day. When this total exposure value was applied to the four mathematical models for the estimation of human risk, between 5 and 560 potential excess lifetime leukemias were predicted for each million persons of the population exposed, or between 750 and 34,000 potential excess lifetime leukemias for this subpopulation.

The remainder of the 1970 U.S. population, approximately 54 million rural dwellers, was considered under Scenario B. Probable exposure occurs by inhalation, ingestion of drinking water and foodstuffs, and the use of gas stations. Average daily exposure was estimated to be 0.3 mg/day benzene. When this value was applied to the risk models, between 3 and 420 potential excess lifetime leukemias were predicted per million people exposed or between 162 and 23,000 potential excess lifetime leukemias for the rural dwellers that comprise this subpopulation.

The third scenario developed, Scenario C, includes residents near a user or manufacturing plant. For this subpopulation, inhalation exposure probably represents a considerably higher benzene intake than for the other two scenarios. Total exposure was estimated to be 0.8 mg/day. The number of individuals comprising this subpopulation cannot

TABLE 7-1. POTENTIAL RISK ESTIMATES FOR BENZENE EXPOSURE SCENARIOS USING DIFFERENT MODELS

	Predicted Number of Excess Lifetime Leukemias per Million Population				
Scenario and Data Base	ADL Linear Model	ADL Log Probit Model	CAG Linear Model	MIT Linear Model	Overall Range
A (0.4 mg/day)					
Aksoy	80	5	224	101	
Infante	120	9	112	560	5-560
B (0.3 mg/day)					
Aksoy	60	3	168	76	
Infante	90	5	84	420	3-420
C (0.8 mg/day)					
Aksoy	160	21	448	202	
Infante	240	32	224	1119	21-1119
Smoking Factor[a] 1.4 mg/day)					
Aksoy	280	60	784	350	
Infante	420	90	392	1960	60-1960

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[a] These numbers may be added to the numbers for the scenarios above.

be accurately determined from available data. A potential of 21 to 1119 potential excess lifetime leukemias per million population exposed were predicted using the several risk models.

The approximately 54 million people who smoke cigarettes in the United States absorb, on average, another 1.4 mg/day benzene (based on consumption of 1.56 packs/day). If no other potential source of benzene existed, benzene in cigarette smoke could contribute between 3200 and 64,000 potential excess lifetime cases of leukemia to U.S. incidence data, or 60 to 1960 potential lifetime cases of leukemia per million population exposed.

Dermal exposure to benzene can occur both occupationally and to residential consumers. Both exposures are probably sporadic and may only occur in a small population. Still, it is possible to absorb a considerable amount of benzene percutaneously.

Although there is a broad range in the numerical estimation of the potential risk of benzene exposure, some conclusions can be drawn. Cigarette smoking (1.56 packs/day) appears to produce a larger exposure to benzene than all of the routes combined in any of the three exposure scenarios. The potential exposures from urban and rural living are similar because inhalation exposure, at ambient air concentrations, is small compared with food ingestion. Data on both food and water, however, are regarded as incomplete, making their contribution to risk somewhat uncertain. Finally, living or working near large atmospheric benzene sources, such as petroleum refineries and chemical plants, could double the benzene exposure and thus the leukemogenic risk. The size of this subpopulation has not been estimated, however.

7.2 BIOTA

A comparison of those levels of benzene in water that have been observed to cause toxic or lethal effects in the laboratory with monitored ambient levels does not indicate a serious cause for concern. Though effluent levels of benzene have exceeded 1 mg/l in some instances, the dispersion effect of flowing water tends to reduce the concentration fairly quickly. Thus, while mobile aquatic populations are not endangered, benthic or algal populations might be subject to chronic exposure and suffer some loss in numbers or health. However, these incidents are highly localized and not significant in number to cause large-scale ecological community effects.

APPENDIX A. VEHICLE RELEASE OF BENZENE

This appendix contains the results of JRB Associates and Arthur D. Little, Inc. (ADL) calculations on evaporative and exhaust emission of benzene from automobiles. The ADL work is based on a set of (SWRI unpublished) exhaust emission factors significantly lower, and more recent, then the JRB figures. Thus, these latter were used in the final materials balance. The reader is referred to the JRB Materials Balance report for greater detail.

1. Automobiles

   A. Evaporative - JRB calculation of 11,000-21,000 kkg/yr

   B. Exhaust

      1) With catalytic converter

         a. JRB emission rate: 0.005-0.020g/mile

Emissions = (8.55 x 10^{11} vehicle miles) (.005-.02g/mile)
= 4275-17,098 kkg

b. ADL data: 0.005-0.007g/mile

Emissions = (8.55 x 10^{11} vehicle miles) (0.005-0.007g/mile)
= 4275-5984 kkg

2) Without catalytic converter

a. JRB data: 0.05-0.15g/mile

Emissions = (3.16 x 10^{11} vehicle miles) (0.05-0.15g/mile)
= 15,810-47,430 kkg

b. ADL data: 0.025-.073

Emissions = (3.16 x 10^{11} vehicle miles) (0.025-0.073g/mile)
= 7905-23,082 kkg

C. Total auto emissions including evaporative

1) JRB: 31,085-85,528 kkg

2) ADL: 23,180-50,066 kkg

2. Motorcycles

A. Crankcase emissions

JRB calculation of 0-73 kkg

B. Evaporative emissions

JRB calculation of 44 kkg

C. Exhaust

1) Two stroke engine
JRB emission rate: 0.27-0.8g/mile

Emissions = (2.31 x 10" vehicle miles) (0.27-0.8g/mile)
= 6247-18,511 kkg

2) Four stroke engine
JRB emission rate: 0.05-0.15g/mile

Emissions = (2.31 x 10" vehicle miles) (0.05-0.15g/mile)
= 1156-3470 kkg

D. Total motorcycle emissions

JRB estimate: 1200-18,628 kkg

3. Trucks and buses

A. Light duty trucks

1) Evaporative

JRB calculation of 4578 kkg

2) Exhausts

JRB emission rate 0.06-0.2g/mile

Emissions = (2.8 x 10" vehicle miles) (0.06-0.2g/mile)
= 16,835-56,116 kkg

3) Total emissions

JRB: 21,413-60,744

B. Heavy trucks and buses

1) Evaporative

B. Heavy trucks and buses

1) Evaporative

JRB calculation: 26 kkg

2) Exhaust

JRB emission rate: 0.2-0.7g/mile

Emissions = 170-595 kkg

3) Total heavy truck and bus releases

JRB: 196-621 kkg

4. Total vehicle releases (including motorcycles, buses and trucks)

A. JRB: 53,894-165,521 kkg

B. ADL· 45,989-130,059 kkg

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APPENDIX B. EXAMS SCENARIOS

1. Petroleum Refinery

    EGD data

        flow - 2 MGD

        [Benzene] in effluent - 60 μg/l

        assume 24 hour day

    Loading Rate = 1.9 g/hr = 0.002 kg/hr

2. Petroleum Refinery

    from EGD data

        assume flow = 10 MGD, 24 hour day

        [Benzene] = 2 mg/l

    Loading Rate = 3.15 kg/hr

3. Solvent Use (textiles industry)

    from EPA's "GAD to Water" 1976

        flow = 3.74 million l/day

        [Benzene] = 64 μg/l

        assume 8 hour day

    Loading Rate = 30 g/hr = 0.03 kg/hr

APPENDIX C. LIQUID CONSUMPTION FOR EXPOSURE ESTIMATES

In 1979, the average per capita soft drink consumption was 35.9 gal/yr or 0.37 l/day. The range was between 28.7 and 47 gal/yr (0.3-0.49 l/day). Total soft drink consumption was given as 7,950,287,700 gallons; thus indicating that the analyst used a U.S. population figure of 221, 456,482. Using this number, the total beverage market sales of 18.2 billions gallons, and the present share of the market segments, per capita consumption of each type of beverage was calculated as shown in Table C-1. The "average" person thus consumes 0.85 l/day of beverages, including sodas, juices, beers, spirits, soft drinks, and bottled waters. (Of course actual individual consumption patterns vary widely.)

Thus, of the typical liquid consumption of 2 ℓ/day, almost half has been commercially prepared. In addition, coffee and tea consumption will reduce the amount of the remaining 1.15ℓ of tap water that is consumed unaltered.

This analysis is important because during commercial preparation, most beverage manufacturers have treated their process water by either ozonation (Westerman 1980), or activated or granular carbon. Both processes are excellent strippers of most organics. Boiling, as in preparation for coffee and tea, also aids in organics removal through evaporation. Therefore, consumption of prepared beverages as a large percent of total liquid intake may considerably reduce the amount of organics consumed, _if_ any are present in the individual's domestic or occupational water supplies.

TABLE C-1. U.S. BEVERAGE CONSUMPTION IN 1979

Beverage	Percent[a] of Market	Total Gallons[b] Consumed	Per Capita Consumption[c] gal/yr	ℓ/day
Soft Drinks	43.8	7,950,287,700[1]	35.9	0.37
Beers	29.2	5,314,400,000	24	0.25
Fruit Drink	12.4	2,256,800,000	10.2	0.11
Soft Drink Mixes	7.8	1,419,600,000	6.4	0.07
Wines	2.5	455,000,000	2.1	0.02
Distilled Spirits	2.5	455,000,000	2.1	0.02
Bottled Waters	1.8	327,600,000	1.5	0.02
TOTAL	100	18,178,687,000	82.2	0.854

[a] Data given in Beverage World (1980).

[b] Total Consumption calculated from total market figure of 18.2 billion gallons and market share.

[c] Per capita consumption based on total consumption and U.S. population of: $\frac{7{,}950{,}287{,}700 \text{ gallons}}{35.7 \text{ gal/person}} = 221{,}456{,}482$ derived from the soft drink data presented in above cited reference.

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